RANDOMIZED CONTROLLED TRIAL BETWEEN LOW DOSE DHAKA REGIMEN AND STANDARD PRITCHARD'S REGIMEN OF MAGNESIUM SULPHATE FOR SEVERE PRE-ECLAMPASIA AND ECLAMPSIA

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BRANCH II

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MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

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CERTIFICATE

This is to certify that the dissertation entitled, **"RANDOMIZED CONTROLLED TRIAL BETWEEN LOW DOSE DHAKA REGIMEN AND STANDARD PRITCHARD'S REGIMEN OF MAGNESIUM SULPHATE FOR SEVERE PREECLAMPASIA AND ECLAMPSIA"** submitted by **Dr. P. BINDU ISAAC,** in partial fulfillment for the award of the degree of Doctor of Medicine in Obstetrics and Gynaecology by the Tamil Nadu Dr. M.G.R. Medical University, Chennai is a bonafide record of the work done by her in the Department of Obstetrics and Gynaecology, Madras Medical College, during the academic year 2006-2009.

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ETHICAL COMMITTEE CERTIFICATE

DATED 25-2-2008

I, Dr. P.BINDU ISAAC apply for the ethical committee certificate for the project **"RANDOMISED CONTROL TRIAL BETWEEN LOW DOSE 'DHAKA' REGIMEN AND PRITCHARD'S REGIMEN OF MAGNESIUM SULFATE IN SEVERE PREECLAMPSIA AND ECLAMPSIA."** Under the guidance of Dr . Latha ,M.D. D.G.O.., Professor ,Institute of Obstetrics and Gynaecology ,Egmore, Chennai-8.

I understand the implications of doing research with human subjects and will fully comply with the regulations and keep the dignity and protect the health of subjects at all costs.

SIGNATURE OF THE POSTGRADUATE STUDENT

I have no objection to guiding this postgraduate student in the project mentioned above.I Shall supervise to the extent that all the human rights are protected and research is carried on with utmost humanitarian principles.

DR. G. LATHA, MD., DGO.,
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SIGNATURE OF THE GUIDE **SEAL OF GUIDE**

I Certify that this project has been presented in front of the Ethical Committee , duly formatted in this institution and that all the members of the ethical committee have given permission to conduct this research.

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CHAIRMAN ETHICAL COMMITTEE

Date : 25.2.2008. Seal

CONTENTS

INTRODUCTION

What makes the BP in pregnancy to rise;

Is still a mystery to many a wise -

How can we find a method of cure?

When the causative factor still remains obscure!

Pre-eclampsia and eclampsia remain one of the serious complications of pregnancy occurring in approximately 7-10 % of pregnant women[1].

According to WHO, it constitutes for about 5 % pregnancies and is responsible for 17.2% of maternal mortality rate and 22% of perinatal mortality rate in India [2].

The principles of management of this condition include control of blood pressure using antihypertensives, control of convulsion, monitoring complications and steps for delivery of fetus[3].

The optimal anticonvulsant for management of severe preeclampsia and eclampsia was disputed till the Eclampsia Collaborative Group published its results in 1995, showing clearly that magnesium sulphate is more efficacious than phenytoin or diazepam in diminishing the risk of further convulsions and also in decreasing maternal and neonatal mortality[4].

The publication of MAGPIE trial in 2002, finally resolved the long standing controversy and uncertainity on whether prophylaxis is useful in preventing the first seizure in severe preeclampsia and that magnesium sulphate was the agent of choice[5].

However in many PHC's and peripheral hospitals magnesium sulphate has still not gained popularity because of fear of the rare but sometimes fatal toxicity of respiratory depression. This led to the search for an optimum dosage regimen for women smaller than their western counterpart.

In 1998, study concluded in Dhaka, developed a low dose (Dhaka) regimen, which appeared to control and prevent convulsions effectively and had a low side effect profile than the Pritchard regimen [6].

To determine whether the low dose regimen has less side effects while at the same time being equally effective as the Pritchard regimen, a randomized clinical trial was carried out.

REVIEW OF LITERATURE

Hypertensive disorders complicating pregnancy are common and form one of the deadly triad, along with haemorrhage and infection, that contribute greatly to maternal morbidity and mortality [7]

Preeclampsia and eclampsia remain a difficult puzzle to solve. It is complex hypertensive disorder of pregnancy affecting multiple systems. Preeclampsia and eclampsia are not distinct disorders but are differentiated according to their clinical symptoms. The mildest disorder in this continuum is gestational hypertension. In preeclampsia hypertension and proteinuria are present, and when convulsions occur in addition to these signs, the condition is referred as eclampsia. Traditionally it was believed that eclampsia evolves in a fairly linear fashion from mild to severe form of preeclampsia to seizures. It is now known that the progression from mild preeclampsia and eclampsia may not occur in all women. In a retrospective study **Katz et al** [8], found that in 60% of cases seizures were the first sign of preeclampsia. However in severe preeclampsia compared to mild preeclampsia, the incidence of eclampsia increases three fold (3% and 1% respectively) [9]

DEFINITION AND CLASSIFICATION OF PREECLAMPSIA

DEFINITION - ACOG

Pre-Eclampsia is defined as a rise in diastolic BP of 90 mm Hg or more or systolic BP of 140 mm Hg or more recorded on at least two occasions six hours apart and the development of Proteinuria of 300 mg/L or more in 24 hours or presence of 1 gm or more per litre at random on at least two occasions six hours apart after 20 weeks of gestation in a previously normotensive nonproteineuric woman and which regresses postpartum.

CLASSIFICATION

1. NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAM WORKING GROUP (2000)

Gestational hypertension

- BP>140/90 mmHg for first time during pregnancy
- No Proteinuria
- BP returns to normal <12 wks postpartum
- Final diagnosis made only postpartum
- May have other signs / symptoms of preeclampsia for example, epigastric discomfort or thrombocytopenia

Preeclampsia

Minimal criteria

- BP >140/90 mmHg after 20 weeks gestation
- Proteinuria >300 mg/24 hrs or >1+ dipstick

Increased certainty of preeclampsia

- BP>160/110 mmHg
- Proteinuria 2.0g/24 hr or >2+ dipstick
- Serum creatinine >1.2 mg, unless known to be previously elevated
- Platelets < 1,00,000/mm^3
- Microangiopathic hemolysis
- Elevated SGOT/SGPT/LDH
- Persistent headache or other cerebral or visual disturbances
- Persistent epigastric pain

Eclampsia

Seizures that cannot be attributed to other causes in a women with preeclampsia

Superimposed preeclampsia on chronic hypertension

- New onset proteinuria 300 mg/24 hr in hypertensive women but no proteinuria before 20 weeks gestation

- A sudden increase in proteinuria or BP or platelet count <1,00,000/mm^3 in women with hypertension and proteinuria before 20 weeks gestation.

Chronic hypertension

- BP >140/90 mmHg before pregnancy or diagnosed before 20 weeks gestation not attributable to gestational trophoblastic disease (or)

- Hypertension first diagnosed after 20 weeks gestation and persistent after 12 weeks postpartum

PROTEINURIA

Proteinuria is an important sign of preeclampsia. It reflects the degree of glomerular damage that causes leakage of proteins through the basement membrane. The amount of proteinuria is used as an indicator for assessing the severity of preeclampsia. Significant proteinuria is

described as 300mg per litre or more of urinary protein loss in 24 hours or persistent 30mg/dl (1+ dipstick) in random clean catch samples on at least 2 occasions collected 6 hours apart. If proteinuria is > 5gm /24 hours or persistent 2+ dipstick or more the condition is labelled as severe pre-eclampsia.

Dipsticks are routinely used to measure proteinuria and the colour changes correspond to

Protein;	Trace	–	0.1 gm/L
	1+	–	0.3 gm/L
	2+	–	1.0 gm/ L
	3+	–	3.0 gm/L
	4+	–	10.0 gm /L

INDICATORS FOR SEVERITY OF PREECLAMPSIA

ABNORMALITY	MILD	SEVERE
Diastolic BP	<100 mm Hg	110mmHg or higher
Proteinuria	Trace to 1	Persistent 2 +or more (at least 5 gm /24 hrs)
Headache	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present(output less than 400 -500 ml)
Convulsion	Absent	Present (Eclampsia)
Serum creatinine	Normal	Elevated
Thrombocytopenia	Absent	present
Elevated liver enzymes	Minimal	Marked
Fetal growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

One or more of the above criteria must be present for the condition to be labelled as severe preeclampsia.

INCIDENCE OF PREECLAMPSIA

The incidence of preeclampsia is 7-10% depending on the population studied and definition of preeclampsia. The incidence of preeclampsia and eclampsia in Institute of Obstetrics and Gynaecology, Chennai is 12–14%.Worldwide approximately 50,000 women are estimated to die annually because of eclampsia. The overall maternal death rate of eclampsia is 2%, but varies geographically according to the quality of area's health care system. Death related to toxemia of pregnancy accounts for 11% of maternal deaths in India[10]. Preeclampsia and eclampsia are also among the major contributors to perinatal mortality and morbidity. The perinatal mortality among babies born to eclamptic mothers was 32.7% compared to 10.5 % for total perinatal mortality[11]. Preeclampsia is strongly associated with IUGR, low birth weight, preterm delivery, respiratory distress syndrome and admission to NICU.

How pregnancy incites or aggravates hypertension remain unknown.Intensive research is ongoing,regarding the risk factors that may predispose and the predictors of this condition and whether prevention is possible by any pharmacological or non pharmacological strategies.

RISK FACTORS FOR PREECLAMPSIA

Certain women have been identified to be at risk for development of hypertensive disorders of pregnancy[12].

RELATIVE RISK FACTORS

GENETIC FACTORS

Genetic predisposition

Ethnicity: more common in Blacks &Asians

Family H/O preeclampsia

Pregnancy by ovum donation

AGE & PARITY

Teenage pregnancy

Age > 40 years

Long interval between pregnancies

Nulliparity

PARTNER RELATED RISK FACTOR

Change of partner

Limited sperm exposure

Donor insemination

Partner who fathered a preeclamptic pregnancy in another woman

Presence of specific underlying disorders

Chronic hypertension

Renal disease

Obesity (body mass index > 35 kg/m2)

Diabetes mellitus

Maternal low birth weight

Polycystic ovarian syndrome

Migraine

Collagen vascular disorders

Uncontrolled hyperthyroidism

Factor V leiden deficiency & thrombophilia

Activated protein C resistence, protein S deficiency

Antiphospholipid antibodies

Hyperhomocysteinemia

Sickle cell disease, sickle cell trait

Women with excessive snoring

Pregnancy related risk factors

Multiple pregnancy

Congenital anomalies

Hydrops fetalis

Chromosomal anomalies (trisomy 13, triploidy)

Hydatiform mole

Urinary tract infection

Exogenous factors

Smoking (risk reduction)

Stress, work related psychological stress

Previous H/O preeclampsia

Raised BP (diastolic >80) at booking

ETIOPATHOGENESIS

The pathophysiology of disease is far from being understood. **Ziefel** described it as 'disease of theories'. Any satisfactory theory concerning the etiology of pathophysiology of preeclampsia must account for the observation that hypertensive disorders due to pregnancy are very much likely to develop in women

i) who are exposed to chorionic villi for first time

ii) who are exposed to superabundance of chorionic villi as with twins and hydatiform mole

iii) have preexisting vascular disease

iv) Are genetically predisposed to hypertension developing during pregnancy

Roberts et al., proposed that maternal endothelial cell dysfunction is the key event resulting in diverse clinical manifestations of preeclampsia and considered preeclampsia as a two stage disease [13] . The initiation of preeclampsia seems to be related to decreased placental perfusion (stage 1) which then results in the maternal syndrome of preeclampsia (stage 2). The maternal syndrome reflects a state of generalized dysfunction secondary to excessive amount of circulating antiendothelial factors such as SFlt – 1[14].

Although chorionic villi are essential, they need not be located within the uterus. A fetus is not a requisite for preeclampsia . Regardless of precipitating etiology, the cascade of events that lead to preeclampsia is characterized by a host of abnormalities that result in vascular endothelial damage with vasospasm, transudation of plasma and ischemic and thrombotic squeal. **(Brunner and Gavras, 1975).**

According to **Sibai (2003)**, currently plausible potential causes include the following

i) Abnormal trophoblastic invasion

Failure of secondary wave of trophoblastic invasion, which mainly occurs at 16-20 wks. This trophoblastic invasion of spiral arterioles is responsible for destruction of muscular layer making vessels lose their refractoriness to vasopressors[15].

ii) Immunological intolerance between maternal and fetoplacental tissues

Risk of hypertensive disorders is enhanced in circumstances where formation of blocking antibodies to antigen sites in

placenta may be impaired as in first pregnancy and multiple pregnancy[16,17]

iii) **Maternal maladaption to cardiovascular or inflammatory changes of normal pregnancy[18]**

iv) **Dietary deficiencies -** deficiency of Zinc, Calcium, Mg, Vit C[19]

v) **Genetic predisposition**

Chesley & Cooper (1986)[20] concluded that preeclampsia is highly heritable. **Wilson & coworkers (2004)** reported 60% concordance in monozygotic female twin pairs. An association with HLA DR4 & Angiotensinogen gene T235 was found to have higher incidence of preeclampsia[21].

MULTISYSTEM EFFECTS OF PREECLAMPSIA

Brain	-	edema, haemorrhage, infarction
Eyes	-	sudden retinal detachment, cortical blindness, papilledema
CVS	-	Hypertension, pulmonary edema
RS	-	Pulmonary edema, aspiration pneumonitis
Liver	-	congestion, hemorrhage, infarction, rupture
Kidney	-	glomeruloendotheliosis, nephrotic syndrome, ARF
Blood	-	thrombocytopenia, microangiopathic hemolytic anemia, DIC
Reproductive	-	IUGR, prematurity, placental abruption, IUD
Skin	-	edema, petechia, ecchymosis
Mucosa	-	laryngeal edema

PREDICTION OF HYPERTENSIVE DISORDERS OF PREGNANCY

A variety of biochemical and biophysical markers primarily on rationales implicated in the pathology and pathophysiology of preeclampsia have been proposed for its prediction, but to utmost disappointment of many workers there is no reliable, valid or economical screening test available for the prediction of preeclampsia **(Friedman and Lindheimer)** [22].

Mid trimester Blood Pressure

The absence of fall in mid trimester blood pressure has been noted in many patients who later on developed preeclampsia. Women with a mid trimester mean arterial pressure >90mm Hg have a three fold risk of developing preeclampsia.

Roll over test

An increase of > 20mm Hg of diastolic BP induced by having women at 28-32 weeks assume the supine position after lying laterally recumbent predicted gestational hypertension with a positive predictive value of 33% **(Gant and Colleagues)**[23].

Handgrip test

Degari et al., found that increase in diastolic BP >20mmHg during a hand grip test at 28-32 weeks is associated with increased incidence of preeclampsia with a sensitivity of 81% and specificity of 68.4%.

Angiotensin II infusion test

Women requiring less than 8ng/kg/min of angiotension II to raise their diastolic BP by 20 mm Hg had a positive predictive value of 20-40% of developing preeclampsia **(Freidman).**

Uric acid

Elevated serum uric acid level due to deceased urate excretion are frequently found in women with preeclampsia. Plasma uric acid value exceeding 5.9 mg/dl at 24 wks has a positive predictive value of 33%. **(Jacobson and Colleagues).**

Urinary calcium excretion

24 hours urinary calcium excretion less than 12mg/dl had a sensitivity of 70% and a positive predictive value of 91% **(Sanchez – Romos)**.

Urinary Kallikrein excretion

Kallikrein is an important regulator of blood pressure and it has been observed that its diminished excretion due to reduced levels in the circulation might precede development of preeclampsia **(Miller et al)**. Urinary kallikrein creatinine ratio at 16 – 20 weeks can be used to predict patients at risk for preeclampsia.

Serum fibronectin

Endothelial cell activation is the likely cause of elevated serum cellular fibronectin levels in some women with preeclampsia. In patients with preeclampsia, a 2 fold increase in fibronectin >400ng/ml is seen.

It has a sensitivity of 69% and positive predictive value of 12 %. **(Pallberg and Colleagues)** [24].

Uterine artery Doppler velocimetry

Measurement of uteroplacental vascular resistance during Doppler ultrasound evaluation of uterine artery impedance in the second trimester has been used as early screening test for preeclampsia **(Bweley)**[25]. **Audibert and coworkers (2005)**[26] combined second trimester maternal serum screening for βHCG and AFP with uterine artery notching and found sensitivity that ranged from 2 – 40%.

PREVENTION OF PREECLAMPSIA - IS IT POSSIBLE?

The therapeutic options available to the patient and her physician once a diagnosis of preeclampsia has been made are very limited. For this reason, much attention has been focussed on strategies for primary prevention and on the identification of subgroups of women with preeclampsia who would benefit from such intervention.

NON PHARMACOLOGICAL PREVENTIVE STRATEGIES

1) Bed rest

Has not been conclusively shown to prevent the development or alter the course of proteinuric hypertension[27] .

2) Dietary sodium restriction

There is no convincing evidence that salt restriction has any role to play in either the prevention or treatment of hypertensive disorders of pregnancy. The physiological volume expansion of uncomplicated pregnancy and the association of chronic hypertension, preeclampsia and intrauterine growth restriction with plasma volumes lower than those measured in normal pregnancy are the common reasons cited for why sodium restriction generally is not recommended to treat hypertension during pregnancy[28].

3) Dietary supplementation

A study by the Dietary Approaches to Stop Hypertension (DASH) Collaborative Group demonstrated that dietary manipulation could significantly lower both systolic and diastolic blood pressure[29].

Zinc and magnesium supplements were tried, though there is no evidence to prove that they prevent preeclampsia [30].

Few non randomized studies showed that supplementation of vitamin C & vitamin E as antioxidant therapy helps in prevention of preeclampsia but confirmation by large scale studies are still needed[31].

Calcium supplementation – initial clinical trials suggested that dietary calcium support during pregnancy is associated with substantial benefit in decreasing the incidence of preeclampsia. Recent studies show no benefit. A recent randomized double masked NICHD trial showed that there was no benefit in calcium supplementation[32].

Fish oil capsules were supplemented, but that again proved ineffective [33].

L- Arginine has been found to be useful in the prevention of preeclampsia, but it was an isolated study and no other studies confirmed its findings[34].

PHARMACOLOGICAL PREVENTIVE STRATEGIES

Diuretics

A review of nine randomized placebo controlled studies involving 7700 women, where diuretics were given to prevent preeclampsia showed no difference in incidence of preeclampsia or perinatal mortality [35].

Antihypertensives

There is no evidence that antihypertensive agents can prevent preeclampsia, although the use of antihypertensive agents in women with

preeclampsia and severe elevation in BP (170/110mmHg) has been shown to prevent cerebrovascular accidents, such treatment does not alter the natural course of the disease[36].

Low dose aspirin

Aspirin blocks production of eicosonoids, by irreversibly inhibiting the action of enzyme cyclooxygenase (COX) which is the rate limiting step in the prostanoid biosynthetic cascade. Thromboxane from platelets produces vasoconstriction and platelet aggregation whereas prostacyclin produced by vascular endothelial cells is vasodilator and inhibits platelet aggregation. An imbalance in favour of vasoconstriction and platelet aggregation (TXA2 > PGI2) has been demonstrated early in pregnancies destined to develop preeclampsia and has been implicated in the pathophysiology.

The platelets lack DNA genome and therefore unable to regenerate COX enzyme, theoretically therefore aspirin should alter the process by tipping the balance in favour of production of PGI2.

Early clinical trials and meta analysis suggested that low dose aspirin can be used with no associated risk to mother and fetus[37]. These reports led to the widespread use of prophylactic aspirin to prevent

preeclampsia. The largest trial to date CLASP study, a multicentre trial incorporating large number of patients, however, suggest that low dose aspirin has very little, if any, effect on the incidence of preeclampsia and may indeed have significant adverse effects (most notably a possible increased risk of abruptio placenta) [38].

Thus to date, no single strategy has proven beneficial for prevention of preeclampsia in either low risk or high-risk population.

Since prediction and prevention of preeclampsia and eclampsia is still far away from becoming a reality, focus is still on treatment of this condition to optimize maternal and fetal outcome.

Basic management objectives of any pregnancy complicated by preeclampsia are:

(i) Termination of pregnancy with the least possible trauma to mother and fetus.

(ii) Birth of an infant who subsequently thrives

(iii) Complete restoration of health to mother

The definitive treatment of women with preeclampsia and eclampsia is delivery. Along with use of antihypertensives to decrease

BP, anticonvulsants are used to prevent occurrence or reduce the recurrence of convulsions in women with severe preeclampsia and eclampsia respectively [39].

OBSTETRIC MANAGEMENT OF SEVERE PREECLAMPSIA AND ECLAMPSIA

The optimal obstetric management of severe preeclampsia and eclampsia is delivery of baby and placenta which alone reverses the condition at term gestation. Depending upon maternal and fetal condition, delivery can be accomplished by induction of labour or caesarean section. The management of severe preeclampsia remote from term will depend on weighing the risks and benefits for mother and baby, after assessing fetal maturity.

ANTIHYPERTENSIVES

Antihypertensive treatment is usually given for diastolic BP >110mm Hg. Goal is to decrease diastolic BP to 90-100 mm Hg.

COMMONLY USED DRUGS

DRUG	DOSAGE	ONSET OF ACTION	DURATION OF ACTION	ADVERSE EFFECTS	MAX DOSE
Parenteral Hydralazine	5-10 mg IV q 20 min	10 -20 min	3-6 hrs	Tachycardia, Headache, Flushing, Aggravation of angina	60 mg
Labetolol	20-80 mg IV q 10 min	5-10 min	3-6 hrs	Flushing, Vomiting, Heart block	300 mg
Sodium nitroprusside	0.25-10µg/ kg/min IV	1 min	1-2 min	Nausea, vomiting, Thiocyanate & Cyanide toxicity	10µg/kg/ minute
Nitroglycerine	5-100 µG/min IV	2-5 min	3-5 min	Anemia, methHb, tachyphylaxis	-
Oral drugs Alpha methly dopa	500 mg PO 8 hrly	2 – 4 hrs	12-24 hrs	Sedation, lethargy Postural hypotension	2 gm
Clonidine	0.2 mg PO	30 min	6-8 hrs	Drowsiness, bradycardia	1.2 mg
Nifedipine	10mg PO q 30 min	10-15 min	4-5 hrs	Headache, syncope tachycardia	120 mg

Nifedipine has become the main stay of treatment for hypertension in pregnancy. It is a member of dipyridine class of calcium channel blockers and is practical, dependable and nonparenteral agent that is easy to administer and useful for acute as well as chronic administration.

Nifedipine limits transmembrane influx of calcium into cardiac and smooth muscles. An unusual characteristic of the drug is that higher the BP, the further the decrease. In humans, nifedipine decreases the BP without any apparent decrease in uteroplaccental blood flow or change in fetal heart rate. Nifedipine and magnesium have synergistic effect causing neuromuscular blockage and hypotension[40, 41].

ANTICONVULSANTS

Magnesium sulphate has been used for treatment and prophylaxis of eclampsia and preeclampsia for more than 70 yrs. Alternatives to magnesium sulphate has been investigated including phenytoin and diazepam. The efficacy of magnesium sulphate in eclampsia and seizure prophylaxis for severe preeclampsia has been well studied and validated[42].

HISTORY OF MAGNESIUM SULPHATE AS ANTICONVULSANT

As early as 1906, magnesium sulphate was injected intrathecally to prevent eclamptic seizures by **Horn** [43].

Rismann (1916) gave the drug subcutaneously

Fischer (1916) gave the drug intravenously

Lazard (1925) popularized the intravenous regimen[44]

There were reports that IM magnesium sulphate controlled convulsions as with tetanus, a similar regimen was used in 1926 by **Dorset** to prevent seizures in women with eclampsia[45].

Eastman & Streptoe (1945)[46] increased the dose of $MgSO_4$ by giving an initial dose of 10gm IM followed by 5gm every 6 hrs.

Pritchard (1955) & Chesley & Tepper (1957) combined the IV with IM dose.

In 1993, the drug was given IV to hundreds of women at Los Angeles General Hospital. In all these studies, the dose of magnesium sulphate was small.

Later **Pritchard[47], Zuspan[48]** introduced IM regimen for magnesium sulphate.

PHARMACOLOGY OF MAGNESIUM SULPHATE

- $MgSO_4$. USP is $MgSO_4.7H_2O$ & not $MgSO_4$
- Its molecular weight is 246.5
- 1 gm of this salt contains 98 mg elemental Mg (10% of total weight)

PHARMACOKINETICS OF MAGNESIUM SULPHATE

The pharmacokinetic profile of magnesium sulphate after IV administration can be described by a two-compartment model with a rapid distribution α phase and a slow β phase of elimination [49].

Normal pregnancy level of Mg is 1.5-2.5 mg/dl.

After administration, about 40% of plasma Mg is protein bound. A loading IV dose of 4-6 gm results in an immediate maternal plasma concentration of 5-9 mg /dl. This increase is transient and it falls to 3-4 mg/dl within 60 minutes. Within 90 minutes about 50% of the Mg moves into bones and other cells[50]. Various authors showed that concentration of Mg in plasma rises gradually after IM injection within 90-120 min, being the usual time to reach the maximum level in plasma[51]. This provided the basis for initiating treatment with IV dose.

Data from **Sibai et al.,** suggests that levels are consistently 1.7mmol/L using a regimen of 1g/hr (used in collaborative eclampsia trial). In contrast, Mg level ranges from 1.7 - 3.3 mmol/L with the 2g/hr maintenance infusion. Pritchard suggested a level of 2 - 3.5 mmol/L and 1.8 -3.0 mmol/L as satisfactory for women with severe preeclampsia and mild preeclampsia respectively. Therapeutic level is between 2-4 mmol/L[52].

Mg is excreted almost exclusively in the urine. About 50% of the infused dose is excreted in urine after 4 hours and 90% of the dose during first 24 hours after an IV infusion of magnesium sulphate. In presence of oliguria or significant renal failure, the maintenance dose should be either decreased or discontinued and Mg level to be monitored carefully.

MECHANISM OF ACTION

The exact mechanism by which Mg protects against seizures has not been established but the prophylactic and therapeutic benefits of Mg are likely due to its ability to counteract vasospasm. Magnesium has both central and peripheral actions.

(i) It causes depression of central nervous system **(Borges and Gucer, 1978).**

(ii) Increase in plasma Mg inhibits acetylcholine release in response to motor nerve impulse, decreases motor end plate sensitivity to acetylcholine and decreases motor end plate potential **(Filmy & Soomjum)**.

These actions do not account for controlling convulsions in eclampsia.

(iii) Magnesium sulphate is an N methyl D aspartate receptor antagonist. Anticonvulsant action of magnesium sulphate is attributed to blocking calcium influx through NMDA – subtype of glutamate channel[53] **(Lipton & Rosenberg 1994).**

(iv) Magnesium sulphate acts by opposing the calcium dependent arterial vasoconstriction. It causes cerebral vasodilatation, and increases cerebral blood flow.

(v) Protects endothelial cells from injury mediated by free radicals.

REPORTED BENEFICIAL EFFECTS

- Decreases systemic vascular resistance & mean arterial pressure. Increases cardiac output **(Cotton, 1984)**
- Increases uterine blood flow **(Harbert, 1969)**
- Increases renal blood flow
- Increases prostacyclin release **(Watson et al)**
- Decreases platelet aggregation **(Watson et al)**
- Decreases plasma renin level
- Decreases angiotensinogen converting enzyme level
- Attenuation of vascular response to vasopressors
- Bronchodilation

REPORTED DETRIMENTAL EFFECTS

- Decreases uterine activity and prolonges labour
- Decreases fetal heart rate variability **(Atkinson, 1994)**
- Increases blood loss after delivery
- Respiratory depression **(Guzman et al)**
- Low apgar score

Magnesium sulphate is the ideal drug with rapid onset of action, non sedative effect on mother and baby, fairly wide safety margin and readily available antidote.

SIDE EFFECTS AND TOXICITY

The effect and toxicity of magnesium sulphate can be linked to its concentration in plasma. The anticonvulsant effects of magnesium in clinically relevant doses do not involve depression of the neuromuscular junction.

The first warning of impending toxicity in mother is loss of patellar reflex at plasma concentration between 9-12mg/dl [51].

Other early signs and symptoms of toxicity include nausea, feeling of warmth, flushing, somnolence, double vision, slurred speech and weakness. Respiratory paralysis occurs at 15-17mg/dl.

Cardiac arrest can be expected at a concentration of 30-35 mg/dl[54]. Careful attention to monitoring guidelines can prevent toxicity. Deep tendon reflexes, respiratory rate, urine output and serum concentrations are most commonly followed valuables with magnesium sulphate use. Laryngeal reflexes are usually intact which protects against aspiration pneumonitis. Injection abscess can occur with IM route.

It is important to keep an ampoule (1gm) 10 ml of 10% calcium gluconate at bedside to be used in case of toxicity.

CONTRAINDICATION

There exists no absolute contraindication except myasthenia gravis and heart block.

EFFECT OF MAGNESIUM ON MOTHER

Effect on cerebrovascular system

Magnesium sulphate is a potent vasodilator, especially in cerebral vasculature and administration of magnesium sulphate to women with preeclampsia decreases intracerebral arterial spasm. Mg both in vivo[55] and in vitro[56] increases production of endothelial vasodilator prostacyclin. Mg also protects against injury by free radicals to endothelial cells in vitro.

Effect on cardiovascular system and respiratory system

Pritchard found variable effect of magnesium sulphate on BP reported that anti hypertensive action was transient[3]. Hypotension has not been constantly produced by magnesium sulphate in management of preeclampsia.

Mg tends to decrease maternal respiratory rate in human subjects. There has been some reports that using magnesium sulfphate as tocolytic might be associated with pulmonary edema[57].

Placental transfer

Mg readily crosses placenta and fetal blood levels correlate well with maternal levels. **Halleuk et al** demonstrated demonstrable Mg level increase in fetal serum within one hour and amniotic fluid within three hours after maternal IV administration[58].

Uterine activity

Stallworthy found transient decrease in frequency of uterine contractions during magnesium sulphate loading dose but no significant change in intensity of uterine contractions[60].

Effect on FHR

The effect on fetal heart rate variability has been a controversial issue, as there is conflicting data in literature[61]. **Atkinson et al** using computerized fetal heart analysis concluded that magnesium sulphate is associated with objectively measured decrease in short term variability but the decrease was not clinically significant. There was no associated decrease in long term variability.

Effect on newborn

An apparent depression in serum calcium levels have been reported in fetuses of mothers treated with magnesium sulphate[62]. Others have reported that Magnesium sulphate treatment does not cause neonatal

hypocalcaemia and that the induced neonatal hypomagnesaemia is resolved within the first 48 hours of life[63]. Although clinical neurological depression has been reported in newborn babies of women with preeclampsia treatment with magnesium sulphate, occurrence of adverse effect on offspring is quite rare[64]. There is no adverse effect on apgar score[65], neonatal mortality or neonatal neurological assessment[66]

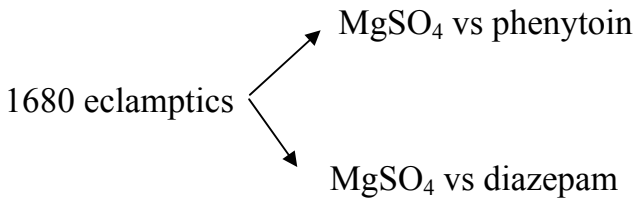
EVIDENCE IN FAVOUR OF MAGNESIUM SULPHATE

Observational studies on subsequent convulsions in women with eclampsia receiving magnesium sulphate.

AUTHOR (YEAR)	NO OF ECLAMPTICS	RECURRENT CONVULSIONS	
		No	%
Pritchard et al (1975)	85	3	3.5
Ge dekoh et al(1981)	52	1	1.9
Pritchard et al[3](1984)	83	10	12.0
Dunn et al (1986)	13	5	38.5
Domisse et al[67](1990)	100	3	3
Sibai et al(1992)	315	41	13.0
All studies	648	63	9.7

These studies paved way for the use of magnesium sulphate in eclampsia for preventing recurrence of convulsions. Various dosage regimens were followed in order to obtain an optimum therapeutic level of magnesium in blood.

In 1995, a study dubbed as the most important obstetric randomized trial of the 20th century showed that, of the three common approaches to controlling eclamptic convulsions, magnesium sulphate was most effective. The **Collaborative eclampsia trial**[4] was a landmark in various aspects. The participation of 1687 women in 27 hospitals in developing countries, achieved more than all the small scale poorly controlled investigations over 50 years, mainly in countries where only 1% of world's cases of eclampsia occur.



Outcomes analyzed were recurrence of convulsions and maternal death. Magnesium sulphate in the trial was administered either IV or IM.

The Collaborative group showed that women allocated $MgSO_4$ had 52% lower risk of recurrent convulsions as compared to diazepam; 67%

decreased risk compared to phenytoin. Maternal mortality was non significant in both groups. Women allocated to $MgSO_4$ were less likely to be ventilated, develop pneumonia or to be admitted in ICU as compared to women who received phenytoin. The babies of women given Magnesium sulphte were less likely to be intubated.

Randomized controlled trials evaluating the use of magnesium sulphate in eclamptics

AUTHOR (YEAR)	INCIDENCE OF RECURRENT SEIZURES	
	MgSO4	OTHERS
Domisse [67](1990)	0/11	4/11
Crowther[68] (1990)	5/24	7/27
Bhalle et al[69](1994)	2/45	11/45
Freidman et al[70] (1995)	0/11	2/13
Collaborative eclampsia trial[4]	60/43	126/452
Collaborative eclampsia trial[4]	22/388	66/387
All studies	88/932	216/935
	9.4%	23%

The above studies further validated the fact that magnesium sulphate was better than other agents (like phenytoin, diazepam) in preventing recurrence of convulsions.

Studies also showed that magnesium sulphate can also be used as prophylaxis in patients with severe preeclampsia to prevent occurrence of convulsions.

The **MAGPIE trial**[5] which involves 10141 women with preeclampsia and their carrers in 175 hospitals in 33 countries proved to be a landmark trial which showed that magnesium sulphate decreases the risk of eclampsia in women with preeclampsia. Women allocated to magnesium sulphate had 58% reduced risk for eclampsia. Women administered magnesium sulphate had 24 % side effects compared to 5 % in placebo group. Although very few side effects were life threatening, most of them were unpleasant and many experienced multiple side effects. Maternal mortality rate was also reduced in women allocated to magnesium sulphate.

Randomized Controlled Trials of Magnesium Sulphate in Severe Preeclampsia

AUTHOR	RATE OF SEIZURES			
	$MgSO_4$		Control	
	No	%	No	%
Odendaal & Hall[71](1996)	2/510	0.4	3/491	0.6
Moodley&Moodley[72](1994	1/112	0.9	0/116	0
Coetzee et al[73](1998)	1/345	0.3	11/340	3.2
Magpie trial group[5](2002)	40/5055	0.8	96/5055	1.9
Belfort et al	7/831	0.8	21/819	2.6
Total	49/6343	0.6	128/6330	2.0

The consistency of results further strengthens the case for magnesium sulphate. The best evidence in generating health care recommendation arises from meta analysis of randomized control trial without heterogenecity .This was validated in a study by **Villar et al, 2004**[74]which compiled the evidence from nine randomized controlled trials and concluded that magnesium sulphate was effective in preventing convulsions in women with eclampsia and severe preeclampsia.

We now have undisputed evidence that magnesium sulphate is useful in women with severe preeclampsia and in eclampsia.

The magpie trial follow up study by **Duley L et al(2007)**[75] has followed up 4782 women originally recruited to the study at the end of two years and concluded that magnesium sulphate was not associated with excess of death or disability for women after two years.

Another study by **Dudley C et al(2007)** [76] has successfully followed up 4483 children born to mothers who were recruited in the Magpie trial at the end of 18 months and concluded that magnesium sulphate was not associated with any clear difference in risk of death or disability for children at 18 months.

LOW DOSE MAGNESIUM SULPHATE

With substantial evidence to say that magnesium sulphate is choice for preeclampsia and eclampsia and its apparent safety, it was hoped that magnesium sulphate would be used invariably in above situation but the fear of fatal toxicity of respiratory depression in women who are much smaller than western women and in institutions where serum Mg monitoring is not feasible, magnesium sulphate is still used with some reluctance.

The next research question obviously was “what is the minimum effective dosage?”

Because of the small size of Bangladesh women and concern about toxicity in circumstances where measuring Mg level would be difficult, a study was conducted in Bangladesh to test the efficacy of low dose regimen of magnesium sulphate.

The objective of study was to record the efficacy of low dose **"Dhaka regimen"** in preventing the recurrence of convulsions in eclamptic patients and to identify whether toxicity occurs with this dose **(Begum et al 2001)**[6]. The study included 65 eclamptics receiving low dose magnesium suphfate regimen - 10gm loading dose followed by 2.5gm IM given 4th hourly for 24 hours after the administration of the first dose. Patients were monitored by observing for respiratory rate, deep tendon reflex and urine output. Findings were matched with serum Mg levels. Range of serum Mg was 1.74- 6 mg/dl. Five patients had diminished knee jerk with serum Mg within the therapeutic range 3.5 mg/dl indicating increased sensitivity of women with smaller BMI. They concluded that half the standard dose of magnesium sulphate appeared to be sufficient to control convulsions. It has also been observed that serum concentration of drug is higher during treatment with maintenance regimen in patients with a lower body volume.

Contrary to this, **Phurapradit et al** [77] found that mean serum Mg levels were significantly lower in women having a weight >70 kg than levels observed in patients with weight <70 kg.

Similar study was conducted in India by **Sardesi Suman et al (2003)**[78] in 1060 women (580 eclamptics & 480 imminent eclampsia) reported that eclamptic convulsions were controlled in 91.93% women and recurrence rate was only 7.84% . It also showed that magnesium sulphate was 98.75% effective as seizure prophylaxis in imminent eclampsia.

Shiva et al(2007)[79] conducted a study in 50 eclamptic women comparing Pritchard regimen versus low dose magnesium sulphate regimen and reported a recurrence rate of only 4%.

Since the introduction of low dose regimen the maternal mortality rate has fallen from i6% to 8% in Bangladesh**(Begum et al 2000)**[80]

Comparison of Low dose regimen with the Pritchard's regimen

Patient outcome	**Sardesi et al Low dose regimen**	**Pritchard et al Std regimen**
Recurrence rate	7.89 %	9.7 %
Maternal mortality	2.63 %	5.2 %
Failure rate	0.18 %	1.5%

These results show that low dose regimen is effective in controlling eclamptic fits and their recurrence, with the added advantage of reduced toxicity both in mother and newborn.

The average maternal weight in India is lower compared to western counterpart (45 kg vs 65 kg). In this situation, it is appropriate to reduce the dose of magnesium sulphate in Indian women because of their lower body weight and thus lower intravascular distribution of drug.

LOADING DOSE OF MAGNESIUM SULphATE

Researchers in Bangladesh with low dose regimen of magnesium sulphate went one step further to test the efficacy of only the loading dose of magnesium sulphate in preventing seizure recurrence in women with eclampsia. 400 women were randomized with either loading dose or low dose regimen of magnesium sulphate. Recurrent convulsions were almost similar in both groups[81].

Patient outcome	**Loading dose N= 202**		**Low dose N= 191**		**P value**
	No	**%**	**No**	**%**	
Recurrent convulsions	8	3.96	7	3.52	ns
Maternal death	9	4.45	10	5.02	ns

AIMS AND OBJECTIVES

OBJECTIVES

To compare Low dose 'Dhaka' regimen vs Standard Pritchard's regimen of magnesium sulphate in patients with severe preeclampsia and eclampsia.

AIMS

(i) To determine if low dose of magnesium sulphate will be sufficient in preventing onset of convulsions in women with severe preeclampsia and prevent recurrence of convulsions in patients with eclampsia.

(ii) To determine if clinical signs and symptoms of magnesium toxicity are less common in women with low dose regimen as compared to Pritchard's regimen.

MATERIALS AND METHODS

TYPE OF STUDY

Randomized controlled trial

PERIOD OF STUDY

August 2007 - August 2008

SETTING

The study was conducted at Institute of Obstetrics and Gynaecology, Chennai. The study was approved by the board of Ethical Committee.

DETERMINATION OF SAMPLE SIZE

The Magpie trial reported that adverse effects in Pritchard regimen of magnesium sulphate was 25% and low dose 'Dhaka' regimen study was nearly 10%. In order to show the magnitude of difference in adverse effects with 95% confidence & 80% power, the sample size in each group was 100 women.

METHODOLOGY

SCREENING

All patients coming to the casualty with a provisional diagnosis of severe preeclampsia or eclampsia were screened for enrollment into the study.

SUBJECT SELECTION CRITERIA

Inclusion criteria

(i) Patients with eclampsia

(ii) Patients with severe preeclampsia with any one of the criteria

- Diastolic BP > 110mmhg ; Proteinuria 2+ and above
- Preeclampsia with symptoms like headache, vomiting, decreased urine output, epigastric pain

Exclusion criteria

(i) Patients having received magnesium sulphate before coming to the hospital.

(ii) Patients with preexisting seizure disorder, heart block or renal failure.

(iii) Postpartum eclampsia with onset of convulsion >72hrs after delivery.

CONSENT

Informed consent in the form of written consent was obtained from the patient or relatives (in situations where patient is indisposed) after explaining the procedure and the drug effects.

Patients were randomly assigned to Group D or Group P using random block number tables. Group D was designated as the study group receiving the low dose (Dhaka) regimen and Group P was designated as the control group receiving the standard (Pritchard) regimen. Drugs including the loading dose and maintenance dose were packed in separate boxes and marked serially as 1-200. Investigator was blinded to identify contents of each box (either Pritchard or low dose regimen).

TREATMENT REGIMEN

GROUP D - Low dose (Dhaka) Regimen :

Loading dose – 4g (20% solution) was given slow IV followed by 3g (50% solution) IM each buttock.

Maintenance dose – 2.5g (50% solution) IM every 4^{th} hourly

GROUP P - Standard dose (Pritchard's) Regimen

Loading dose - 4g (20% solution) was given slow IV followed by 5g (50% solution) deep IM each buttock

Maintenance dose – 5g (50% solution) IM every 4th hourly

A detailed history was obtained from the patients. General examination and obstetric examination was done. Baseline parameters like height, weight, pulse rate, BP, respiratory rate, gestational age, fetal heart rate, bishop's score and adequacy of pelvis were recorded. Initial resuscitation measures were done and blood sample was obtained for routine investigation (as per hospital practice/ guidelines) such as Hb, haematocrit, platelet count, S.bilurubin, SGOT, SGPT, S.urea, S.creatinine, S.uric acid, S.Fibrinogen and S.Electrolytes. Urinary bladder was catheterized and urine albumin recorded using dipstick method.

PROTOCOL FOR ECLAMPSIA

Patient was shifted to ICU and loading dose according to the treatment group assigned was given. Loading dose was administered irrespective of the urine output. Maintenance dose was continued for up to 24 hours postpartum or till 24 hours after the last seizure which ever

was later. Pregnancy was terminated in all cases of eclampsia. Convulsions occurring 30 minutes after the loading dose or at any time later will be treated as recurrent convulsions. An additional dose of 2gm magnesium sulphate was given slow IV for recurrent convulsions.

PROTOCOL FOR IMMINENT ECLAMSPSIA

Patient was administered the loading dose and maintenance dose according to the treatment group assigned. Maintenance dose was continued for 24 hours after the first dose. Decision regarding termination of pregnancy was done depending on the severity of the disease and maturity of the fetus.

Antihypertensives in labour

If women were on antihypertensive, same was continued in labour. If diastolic BP >100mm Hg , Nifedipine 10mg was given 6^{th} hourly.

All patients were monitored on clinical criteria. Hourly monitoring of pulse rate, BP, respiratory rate, urine output, deep tendon reflexes was done.

Maintenence dose of magnesium sulphate was withheld if signs of toxicity in the form of loss of deep tendon reflex, respiratory rate<16/min

and urine output (should be 25ml/hr or 100ml for last four hours) was found. If urine output was less than 25 ml/hr, dose was withheld and fluid challenge given. If urine output was adequate after the fluid challenge, dosage schedule continued. 10 ml of 10% calcium gluconate was kept ready in case of signs of toxicity.

Progress of labour monitored using partogram. Fetal heart rate monitored using intermittent auscultation or electronic fetal monitoring. Decision about optimal mode of termination of pregnancy was done by the consultants of respective units.

OUTCOME

(i) Recurrence of convulsions in eclamptic patients.

(ii) Occurrence of convulsion in preeclampsia patients.

(iii) Side effects like flushing, nausea, vomiting, headache, thirst, drowsiness, induration, abscess.

(iv) Toxicity like absence of deep tendon reflexes, respiratory depression.

(v) Effects of magnesium sulphate on newborn.

RESULTS AND ANALYSIS

This is a randomized trial comparing the efficacy of Low dose (Dhaka) regimen of magnesium sulphate with the Standard Pritchard's regimen in patients with severe preeclampsia and eclampsia, conducted at the Institute of Obstetrics and Gynecology, Chennai during the period of April 2007 - April 2008

200 women with severe preeclampsia and eclampsia were randomized to receive either the low dose regimen or the standard dose regimen. The results were subjected to statistical analysis using chi-square test.

Table - 1

AGE DISTRIBUTION

Age in yrs	LOW DOSE (n = 100)	STANDARD (n = 100)
< 20 yrs	6	7
20 - 30 yrs	91	84
≥ 31 yrs	3	9
Mean age	23.81± 3.64	23.92 ± 4.37

(n= NO OF PATIENTS)

Both the groups were similarly matched with respect to their age group. The mean age group in Low dose regimen and Standard dose regimen was 23.81± 4.37 and 23.92± 3.64 respectively.

Table - 2
PARITY

PARITY	LOW DOSE (n =100)	STANDARD (n=100)
PRIMI	63	65
G 2	24	23
G3	10	8
G4	3	2
G5	-	1
G 6	-	1

(n= NO OF PATIENTS)

Both the groups were similarly matched with respect to parity. Primis constituted 63 % cases in the Low dose group and 65% in Standard dose group.

Table – 3
BOOKING

BOOKING	LOW DOSE (n = 100)	STANDARD (n = 100)
BOOKED IN IOG	15	10
BOOKED OUTSIDE	67	70
UNBOOKED	18	20

Only 15 % cases in Low dose regimen and 10% cases in Standard dose regimen were booked in our hospital.

Table – 4
EDUCATION

EDUCATION	LOW DOSE (n =100)	STANDARD (n =100)
< V	18	24
V - X	54	48
> X	28	28

(n= NO OF PATIENTS)

Table - 5
RELIGION

RELIGION	LOW DOSE (n = 100)	STANDARD (n =100)
HINDU	89	91
MUSLIM	4	2
CHRISTIAN	7	7

TABLE - 6
BODY MASS INDEX

BODY MASS INDEX	LOW DOSE REGIMEN	STANDARD REGIMEN
MEAN BMI	26.20 ± 5.21	25.82±5.06

Both the groups were similarly matched with respect to education, religion & BMI.

The mean BMI in Low dose was 26.20 ± 5.21 and Standard dose was 25.82 ± 5.06 respectively.

Table - 7

BLOOD PRESSURE PARA METERS	LOW DOSE (n =100)	STANDARD (n =100)
DIASTOLIC BP >110mmHg	79	74
ANTIHYPERTENSIVES IN PREGNANCY	40	45

(n= NO OF PATIENTS)

- Both the groups were similarly matched with respect to their Diastolic BP

- 79 % patients in Low dose group and 74 % patients in Standard group had diastolic BP > 110mm Hg.

- 40% patients in Low dose group and 45% patients in Standard regimen group were on antihypertensive therapy.

Table -8
IMMINENT SYMPTOMS

SYMPTOMS OF IMMINENT ECLAMPSIA	LOW DOSE (n =100)	STANDARD (n =100)
HEADACHE	34	39
VOMITING	14	16
EPIGASTRIC PAIN	2	4
BLURRING OF VISION	9	6
DECREASED URINE OUTPUT	14	4

(n= NO OF PATIENTS)

- Most common imminent symptom was headache (34% in Low dose group compared to 39 % in Standard dose group) followed by vomiting (14 % vs 16 %).

- 14% patients in Low dose group and 4% patients in Standard dose group had H/O decreased urine output. Loading dose of magnesium sulphate was given irrespective of urine output. The remaining doses were given if urine output was normal after fluid challenge .

Table -9
RECURRENCE OF CONVULSION IN ECLAMPSIA

	LOW DOSE (n=100)	STANDARD (n=100)
NO OF ECLAMPTICS WHO HAD RECURRENT CONVULSIONS	2	1
NO OF EPISODES	1	1
ADDITIONAL $MgSO_4$ GIVEN	2gm	2gm

P value > 0.05 - Not Significant.

- Of the 41 eclamptic women recruited to the trial, 3 developed recurrent seizures during the treatment period.
- 2 patients in Low dose & 1 patient in Standard dose group.
- All the 3 patients developed recurrent seizures in-between the dosage schedule.
- These patients had only one episode of recurrence which was controlled with 2 gm magnesium sulphate given IV.
- $MgSO_4$ produced effective seizure control in 98 % in Low dose group and 99 % of patients in Standard dose group.
- Recurrence rate of convulsions was 2% in Low dose group and 1% in Standard dose group

Table – 10

OCCURRENCE OF CONVULSION IN SEVERE PREECLAMPSIA

	LOW DOSE (n=100)	STANDARD (n=100)
NO OF PATIENTS WITH SEVERE PREECLAMPSIA WHO HAD CONVULSIONS	1	1
NO OF EPISODES	1	1
ADDITIONAL $MgSO_4$ GIVEN	2gm	2gm

- Of the 159 severe preeclamptic patients recruited to trial, 2 patients developed seizures during the treatment period.

- 1 patient in Standard dose group developed seizures 2 hours after completion of dosage schedule & 1 patient in Low dose group developed seizures during the dosage schedule.

- The seizures were effectively treated with 2 gm magnesium sulphate.

- Magnesium Sulphate was effective as seizure prophylaxis in 99% in both groups.

- Low dose magnesium sulphate was effective as Standard dose in seizure prophylaxis in severe preeclampsia.

Table – 11

SIDE EFFECTS

SIDE EFFECTS	LOW DOSE		STANDARD		P VALUE
	NO	%	NO	%	
FLUSHING	69/92	75	72/94	76.6	0.93
NAUSEA/ VOMITING	10/92	10.9	15/94	15.9	0.42
MUSCLE WEAKNESS	11/92	11.9	10/94	10.6	0.79
THIRST	22/92	30.4	20/94	27.6	0.85
DROWSINESS/ DIZZINESS	10/92	10.9	10/94	10.6	0.95
PAIN/BURNING	50/92	54.3	58/94	61.7	0.38
INDURATION	20/100	20	26/100	26	0.40
ABSCESS	0/100		1/100	1	1.00

SERIOUS MATERNAL TOXICITY	LOW DOSE	STANDARD
RESPIRATORY DISTRESS	0	0
CARDIORESPIRATORY ARREST	0	0

P value >0.05 -Not significant

- 70% patients in Low dose group and 75% of patients in Standard group had at least one side effect.

- 8% patients in Low dose group and 6% patients in Standard group were not able to evaluate early side effects because of low Glasgow coma scale.

- The most common side effect was flushing (75% in low dose group and 76.6% in standard group).
- Pain was another common side effect because the maintenance dose was given IM (54.3% in Low Dose and 61.7% in Standard dose).
- 10.9% patients in low dose group and 15.9% patient in standard group developed vomiting after the administration of magnesium sulphate.
- 1 patient in standard group had injection abscess.
- None of the patients had any serious maternal toxicity like respiratory distress.

TABLE - 12

REASON FOR WITHOLDING MAGNESIUM SULPHATE

REASON FOR WITHHOLDING $MgSO_4$	LOW DOSE (n=100)	STANDARD (n=100)	P VALUE
LOSS OF DEEP TENDON REFLEX	3	8	0.21
OLIGURIA	2	4	0.61
BP NORMALISED	0	1	1.0
CORTICAL VEIN THROMBOSIS	0	1	1.0

P value >0.05 Not significant

- 5 % patients in Low dose group and 14 % of patients in Standard group needed dose deferral.

- Most common reason for withholding $MgSO_4$ was loss of deep tendon reflex (3 % in Low dose group and 8% in Standard group)

- 2% patients in Low dose group and 4% patients in Standard group needed dose deferral due to oliguria inspite of fluid challenge.

- 1 patient in Standard group had cortical vein thrombosis. This patient was shifted to intensive medical care unit for further management.

TABLE - 13

COMPLICATIONS

COMPLICATIONS	LOWDOSE (n=100)	STANDARD (n=100)
VENTILATOR	2	3
HELLP	0	3
RENAL FAILURE	1	1
CORTICAL VEIN THROMBOSIS	0	1
PPH REQUIRING BLOOD TRANSFUSION	1	2

P value >0.05 – Not significant

- 2 patients in Low dose group and 3 patients in Standard group were put on ventilator due to low Glasgow score.
- 3 patients in Standard group had HELLP syndrome. These patients were treated with platelets and Inj. Betamethasone
- 2 patients in Standard group and 1 patient in Low dose group needed blood transfusion for postpartum hemorrhage
- We had one maternal death due to cortical vein thrombosis.

TABLE - 14

MODE OF DELIVERY

	LOWDOSE (n=100)	STANDARD (n=100)	P VALUE
VAGINAL DELIVERY	44	42	NS
CAESAREAN SECTION(CS)	53	55	NS
MANUAL REMOVAL OF PLACENTA	0	1	NS

P -VALUE (NS-Not significant)

- 44 % of patients in Low dose group and 42 % of patients in Standard group delivered vaginally.

- The caesarean section rate was 53 % & 55 % in Low dose group and Standard group respectively.

- 1 patient in Standard group had manual removal of placenta.

TABLE - 15

INDICATION FOR CAESAREAN SECTION

INDICATION FOR CS	LOW DOSE	STANDARD
FETAL DISTRESS	18	24
FAILURE TO PROGRESS	7	5
ABRUPTION	4	4
PREVIOUS LSCS	8	6
BREECH	2	-
FAILED INDUCTION	4	6
LOW LYING PLACENTA	2	-
DETERIORATING MATERNAL CONDITION	4	5
MULTIPLE PREGNANCY	-	1
UNFAVOURABLE CERVIX	4	4

P Value - not significant

- Most common indication for caesarean section was fetal distress in both groups (24 % in standard group compared to 18% in low dose group).

- Failed induction & failure to progress were comparable in both arms. Hence higher dose of $MgSO_4$ was not found to have tocolytic effect.

- 6 patients had postpartum eclampsia (3 in each arm). All the patients had delivered vaginally - hence labor and delivery criteria's were not analyzed for these patients.

TABLE – 16

PERINATAL OUTCOME

BABY DETAILS	LOW DOSE	STANDARD	P VALUE
BIRTH WEIGHT	2.06± 0.76	2.05 ± 0.77	-
STILL BIRTH	20	24	0.61
NEONATAL DEATH	5	4	0.95
REQUIRING NICU CARE	29	34	0.52
RESPIRATORY DISTRESS	14	19	0.45
HYPOTONIA	12	23	0.06
APGAR < 7			
1 MIN	30	44	0.06
5 MIN	12	16	0.54

P value > 0.05 - Not significant

- The mean birth weight in 2.06 ± 0.76 in Low dose group and Standard group was 2.05 ± 0.77.

- The still birth rate was almost similar in both arms (20 % in low dose group and 24% Standard group).

- Most of the still births were those occurring in women whose babies were nonsalvagable (weight < 1 kg / gestational age < 28 wks). Termination was done by vaginal PGE 1(misoprostol).

- 29 % in Low dose group and 34 % babies in Standard group needed NICU care .

- A higher number of neonates had hypotonia in Standard group than the Low dose group (23% vs 12%), but the result was not statistically significant.

- 30% of babies in Low dose group and 44% babies in Standard group had one minute apgar < 7.

TABLE – 17

POSTNATAL DATA

POSTNATAL DATA	LOW DOSE (n=100)	STANDARD (n=100)	P VALUE
PATIENTS ON ANTIHYPERTENSIVE DURING DISCHARGE	16	15	NS
DURATION OF STAY	10.87 ±5.04	10.51±4.83	NS

P value - NS - Not significant

- 16 % patients in Low dose group and 15 % of patients in Standard group were on antihypertensives during discharge.

- The average duration of stay for patients in Low dose group was 10.87± 5.04 and in Standard group was 10.51 ± 4.83 .

DISCUSSION

There is ample evidence to show that magnesium sulphate is the anticonvulsant of choice in women with severe preeclampsia to prevent onset of convulsions and to reduce the number of seizures in patients with eclampsia.

Inspite of various evidences proving magnesium sulphate to be safer drug, it is still used with reluctance in peripheral hospitals because of the fear of magnesium toxicity in the form of occasional respiratory depression in women who are much smaller than western women and where monitoring for serum Mg level is difficult.

This study was conducted to see if a lower dose of magnesium sulphate will suffice in our women in reducing the occurrence or recurrence of convulsions and to see if side effect and toxicity of Mg was less with the lower dose.

The study was a randomized double controlled trial conducted in a tertiary institute. 200 women with severe preeclampsia and eclampsia were randomized to receive either the low dose (Dhaka) regimen or Pritchard,s dose regimen for magnesium sulphate. The randomization was done using a Tippet table. The researcher, patient & medical personnel administering the drug and assessing the side effects were blinded to the contents of the pack (either Low dose /Pritchard's).

The results of this study are discussed as follows:

In comparison of Dhaka regimen with Pritchard's , the groups generated by randomization were well balanced.

In the study by **Sardesi Suman et al** assessing the efficacy of Low dose regimen of $MgSO_4$ among eclamptic and preeclamptic women, majority of patients were in the age group 20 -30 years. In our study too majority of our patients were in the same age group **(TABLE - 1).**

At trial entry 64 % of women were in their first pregnancy comparable to study by **Sardesi Suman et al** where 66.3% of women were in their first pregnancy. **(TABLE -2)**

In the study by **Suman et al** the average weight of women in the trial group was 48.4 kg. In our study the average weight of women was 50.2kg **(TABLE-3)**.

Among the imminent symptoms headache was the most common symptom (39 % in Standard group vs 34% in Low dose group) followed by vomiting (16 % vs 14 %) (**TABLE -8).** This is comparable to study by **Suman et al** which showed headache (54.57%) & vomiting (26.88%) to be the most common imminent symptoms .

Pritchard et al and Sibai showed a recurrence rate of 10 -20 using the Pritchard's regimen.

Collaborative Eclampsia trial showed a recurrence rate of 5.7– 13.2%.

In this study **(Table 9)** the convulsions were effectively controlled in 99% in Standard regimen and 98 % in Low dose regimen.

The recurrence rate of seizures using low dose regimen was 2% in this study. All the three patients had only one recurrent convulsion which was controlled with 2 gm of Mgso4.

This proved that Low dose regimen was as effective as Standard regimen in seizure control in eclamptics, though the results were not statistically significant.

The seizure control rate using Low dose regimen from various studies were comparable to our study

AUTHOR (YEAR)	**Convulsions controlled effectively**	**Recurrence rate**
Sardesi Suman et al (2003)	91.93%	7.89%
Begum et al (2001)	98.46%	1.5%
Shiva et al (2007)	96 %	4%
Our study **(Table - 9)**	98%	2%

In this study**, (Table 10)** among the patients with severe preeclampsia Low dose Mgso4 proved to be a effective seizure prophylaxis in 99 % of patients similar to that in Standard regimen. The failure of prophylaxis was 1 %.

Sardesi Suman et al showed Low dose $MgSO_4$ to be 98 .75 % effective as seizure prophylaxis. The failure of prophylaxis was 1.25 %.

In our study **(Table 11)** 75% of patients in standard dose group and 70% patients in Low dose group experienced at least one unpleasant side effect. This was in contrast to **the MAGPIE TRIAL** which showed only 25 % side effects for $MgSO_4$ compared to 5 % in placebo arm.

The side effects were relatively low in the Low dose group in this study though it was not statistically significant.

The side effect profile in both arms were similar

The most common side effect was flushing which was comparable to side effect profile in **MAGPIE TRIAL.**

As the maintenance dose of Mgso4 was administered IM, pain and induration at the site of injection was noted.

1% patient in Standard group and none in Low dose group had injection abscess, which was reported in the **Collaborative Eclampsia Trial.**

In our study **(Table 12)** Magnesium sulphate was withheld in 14% patients in Standard dose group and 5 % in Low dose group.

Most common cause for dose deferral was loss of deep tendon reflexes (8% in Standard group and 3 % in Low dose group). Though the dose deferral was high in the Standard group, the result was not statistically significant.

The toxicity profile of this study was comparable to two studies :

1) In a study by **Shiva et al (2007)** 32% needed dose deferral due to loss of deep tendon reflexes in Standard group as compared to 8% in Low dose group.

2) In a study by **Begum et al (2001)** 9% needed dose deferral due to Loss of deep tendon reflex using Low dose regimen.

None of the patient had respiratory depression since strict clinical criteria for withholding MgSO4 was used. None of the patients needed calcium gluconate. This was comparable to studies by **Begum et al (2001) & Shiva et al (2000)** using Low dose regimen.

42% women in standard group and 44 % women in Low dose regimen delivered vaginally (**Table – 15).**

Caesarean section rate in this study was 55 % in Standard group & 53% in Low dose group. The most common indication was fetal distress in both arms.

The indication for caesarean section **(Table -16)** was similar in both arms. This showed that there was no apparent tocolytic effect of $MgSO_4$ due to higher dose.

The still birth rate **(Table – 17)** in this study was 24% in Standard group and 20% in Low dose group. Most of the still births occurred in fetuses considered nonsalvagable in our hospital (weight < 1 kg or gestational age < 28 weeks). Termination was done using misoprostol. This was comparable to study by **Shiva et al (2007)** which showed a still birth rate of 32% in Standard group compared to 28 % in Low dose group.

In this study a higher number of neonates in Standard group had hypotonia (23%) when compared to Low dose group where 12 % of neonates had hypotonia but this was not statistically significant. This was comparable to study by **Shiva et al (2007)** which showed 28% neonates in standard group had hypotonia compared to 4% in low dose group.

The sample size in this study may have been insufficient to detect minor difference which exists between the two drug regimen in terms of incidence of side effects or recurrence of convulsions, but these minor differences may not have clinical implications .

The incidence of recurrent convulsions in eclamptic women and occurrence of convulsions in severe preeclamptic women were comparable in both Pritchard's and Dhaka regimen. From this study low dose regimen may suffice in our patients but the sample size is too small to bring out minor differences.

SUMMARY

- Majority of women recruited to the study were in age group of 20 - 30 years.
- 65% patients in Standard dose group and 63% patients in Low dose group were primi gravida.
- Low dose magnesium sulphate was effective in preventing recurrence of convulsions in 98% of patients. Recurrence rate of convulsions using low dose regimen was 2%.
- Low dose regimen was effective as seizure prophylaxis in 99% patients. Failure of prophylaxis using low dose regimen was 1%.
- 75% in Pritchard's regimen and 70% patients in Dhaka regimen experienced side effects. Most common side effect was flushing.
- None of the patient recruited had any serious maternal toxicity like respiratory depression.
- 14% patients in Pritchard's regimen and 5% patients in Dhaka regimen needed dose deferral. Most common cause of dose deferral was loss of deep tendon reflex.
- 23% babies in Pritchard's regimen and 12% babies in Dhaka regimen had hypotonia. 34% babies in Pritchard's regimen and 29% babies in Dhaka regimen needed NICU care.

CONCLUSION

This study comparing the efficacy of a low dose 'Dhaka' regimen of magnesium sulphate to the standard Pritchard's regimen showed that

1) Magnesium sulphate is a safe anticonvulsant with very less toxicity and side effect. Side effect profile of Low dose 'Dhaka' regimen were similar to the Standard Pritchard's regimen.

2) Low dose regimen of magnesium sulphate is effective in preventing the recurrence of convulsion in women with eclampsia and preventing occurrence of convulsion in women with severe preeclampsia and the efficacy is similar to that of the standard Pritchard's regimen.

3) Low dose regimen is a cost effective regimen which can be safely used in peripheral hospitals and public health centres .

4) Implication for further research would be to test what the minimum effective dose would be and to see if only loading dose is sufficient.

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PROFORMA

NAME **:** **IP NO. :**

AGE **:**

TYPE OF ECLAMPSIA **;**

TYPE OF REGIMEN **:**

DOA **:**

RELIGION **:**

EDUCATION **:**

OBSTETRIC HISTORY

a) Total no of pregnancies :

b) Any preeclampsia in prior pregnancy:

c) Outcome of last pregnancy :

PRESENT PREGNANCY:

a) Last Menstrual Period : Expected due date :

b) Period of gestation

c) Prior Antenatal visits :

d) No of Antenatal visits :

e) Any high BP in prior visits :

f) Any treatment taken :

g) Any other medical complication :

h) Any treatment taken for medical disease:

ECLAMPSIA DETAILS:

a) Type of eclampsia: b) Date :

c) Time: d) Place:

e) No of episodes: f) Any treatment before admission:

EXAMINATION AT ADMISSION:

a) General condition: b) Height c) Weight

d) BMI: e) Pulse rate: f) BP:

g) CVS & RS: h) Pallor: i) edema:

j) jaundice:

OBSTETRIC EXAMINATION:

a) Per abdomen :

b) Per vaginal :

LABOUR:

a) Onset- i)spontaneous ii)induced

b) Method of induction:

c) Mode of delivery :

d) Indication for CS:

e) Outcome :

COMPLICATION DURING LABOUR/ DELIVERY :

a) Prolonged labour b) Abruption

c) PPH d) Rtained placenta

e) Genital injury f) Postpartum shock

INVESTIGATION :

a) Hb: b) PCV: c) Platelets:

d) Urea: e) Creatinine: f) SGOT:

g) SGPT: h) Electrolytes: i) Urine albumin :

j) Fundus :

NEONATAL RECORD:

a) Sex : b) Birth weight: c) Maturity:

e) Apgar score: f) congenital anomalies:

g) Condition of baby at birth:

CONDITION AT DISCHARGE :

a) Total dose of $MgSO_4$ given:

b) No of recurrent convulsions

c) Was any other drug given to control convulsions:

d) Was the arranged regimen changed due to any reason:

e) If yes, specify reason:

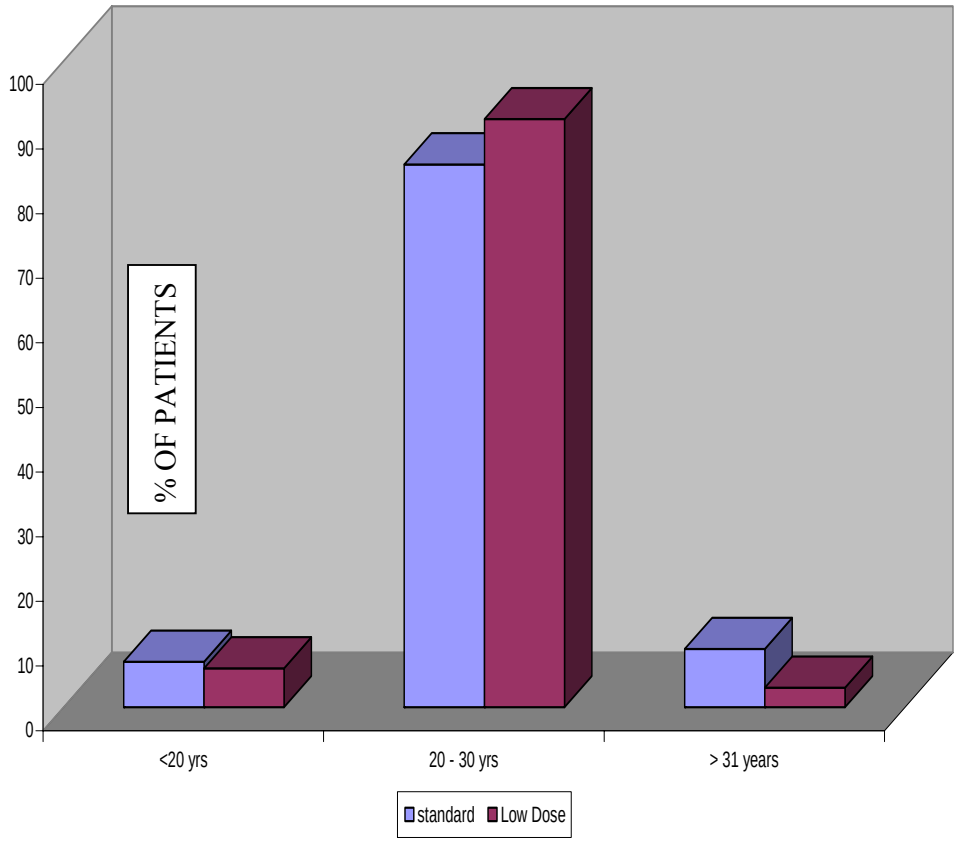
f) Did mother have any signs of toxicity:

GLOSSARY

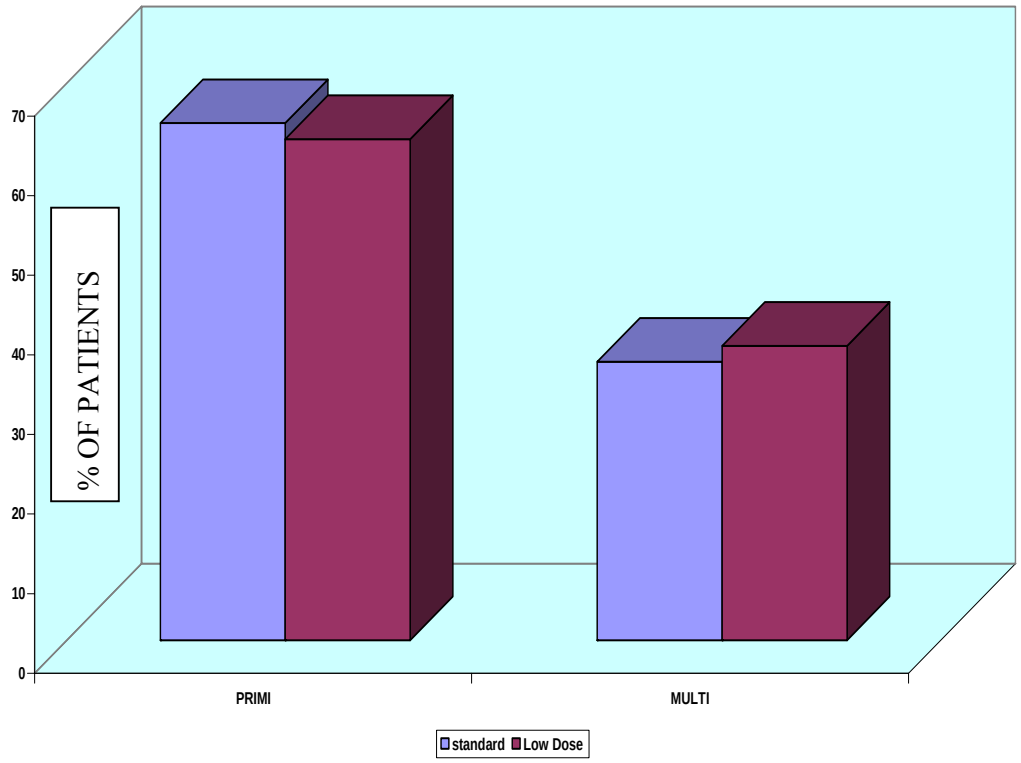
Mg	-	Magnesium
AFP	-	Alpha Feto Protein
BMI	-	Body Mass Index
BP	-	Blood Pressure
β HCG	-	Beta Human Chorionic Gonodotropin
CS	-	Caesarean section
IUGR	-	Intrauterine growth Retardation
LDH	-	Lactate Dehydrogenase
$MgSO_4$	-	Magnesium sulphate
NICU	-	Neonatal intensive Care Unit
PCV	-	Packed Cell volume
PPH	-	Postpartum Haemorrhage
SGOT	-	Serum Glutamate Oxaloacetate Transaminase
SGPT	-	Serum Glutamate Pyruvate Transaminase

FIGURE– 1

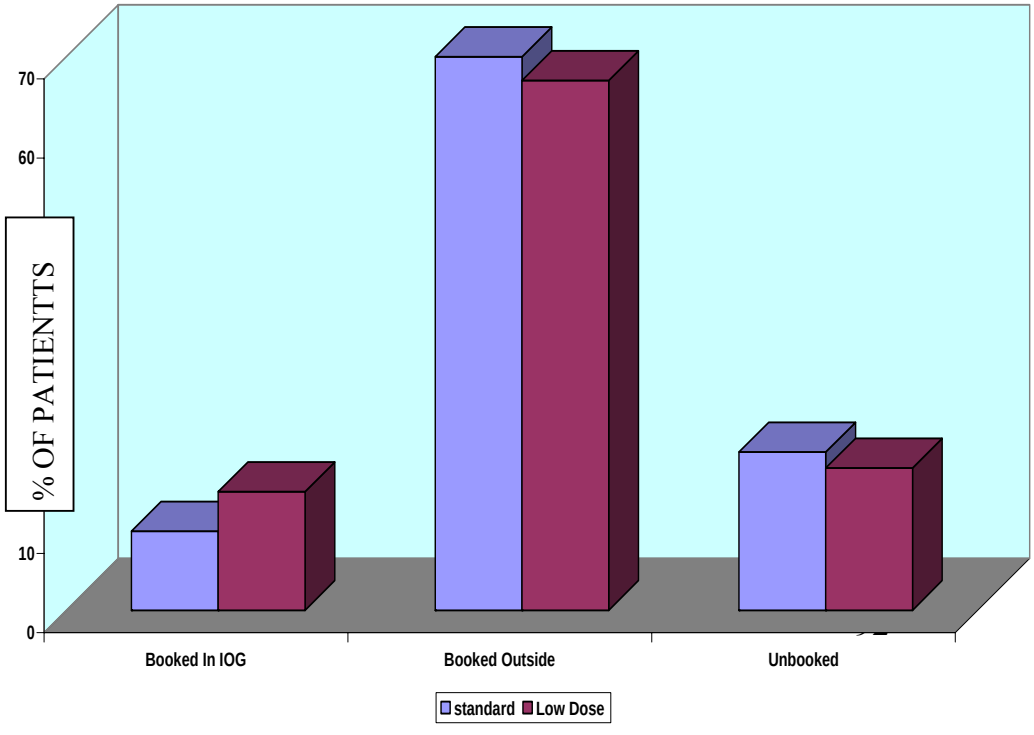
AGE DISTRIBUTION



PARITY



BOOKING



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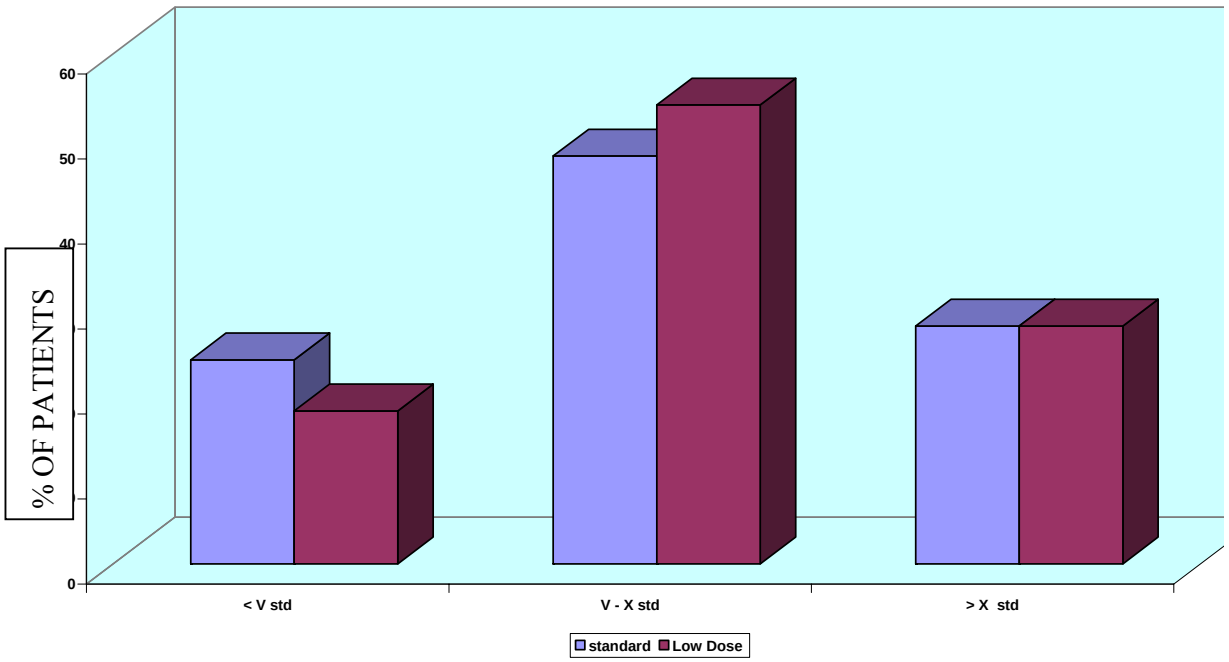
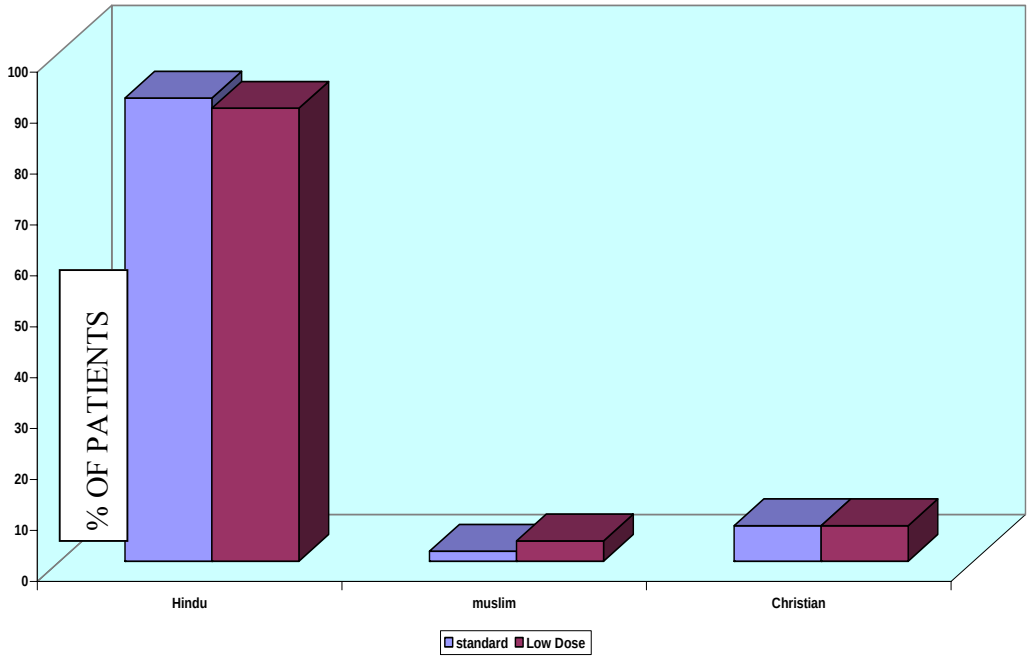
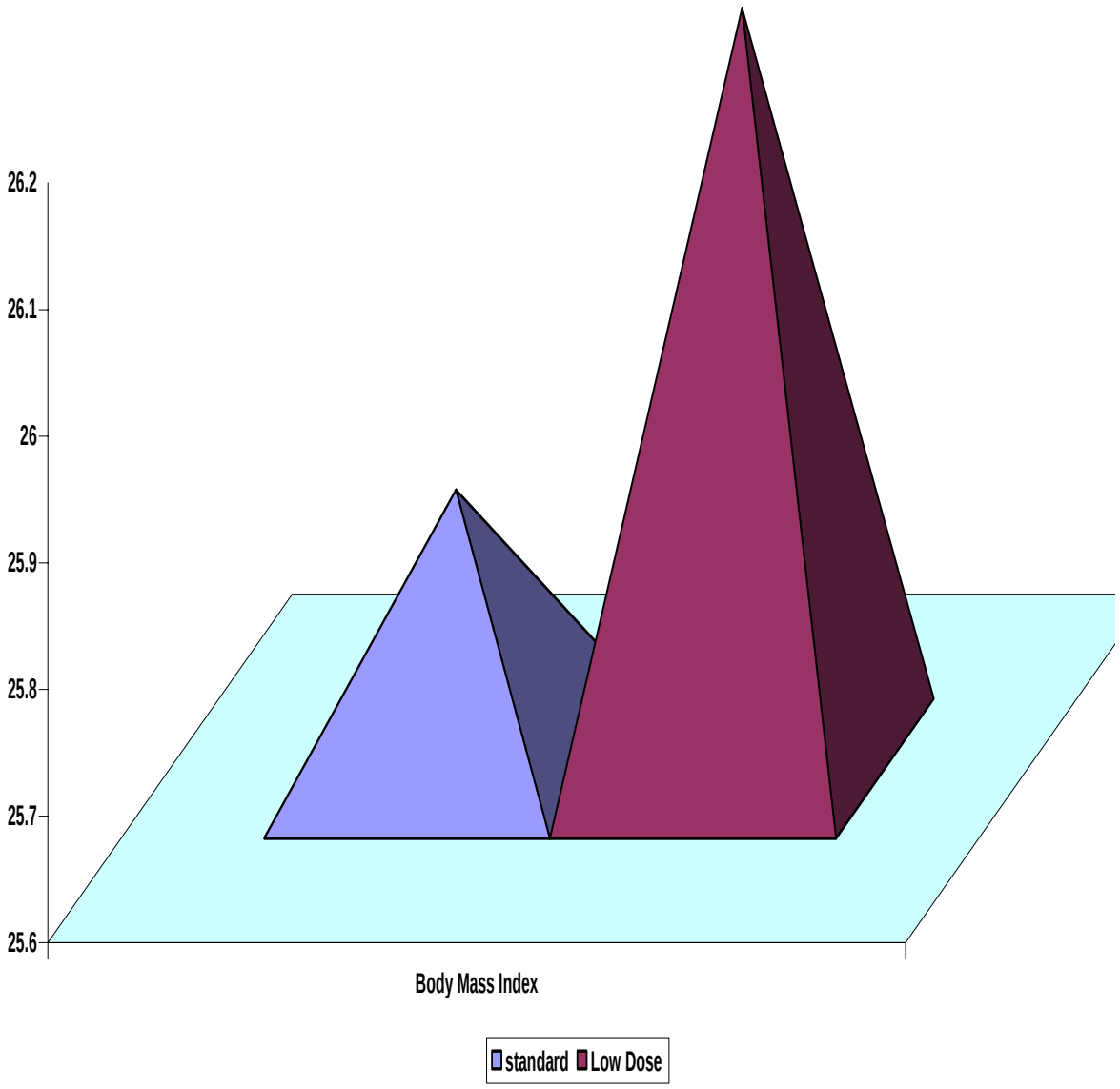


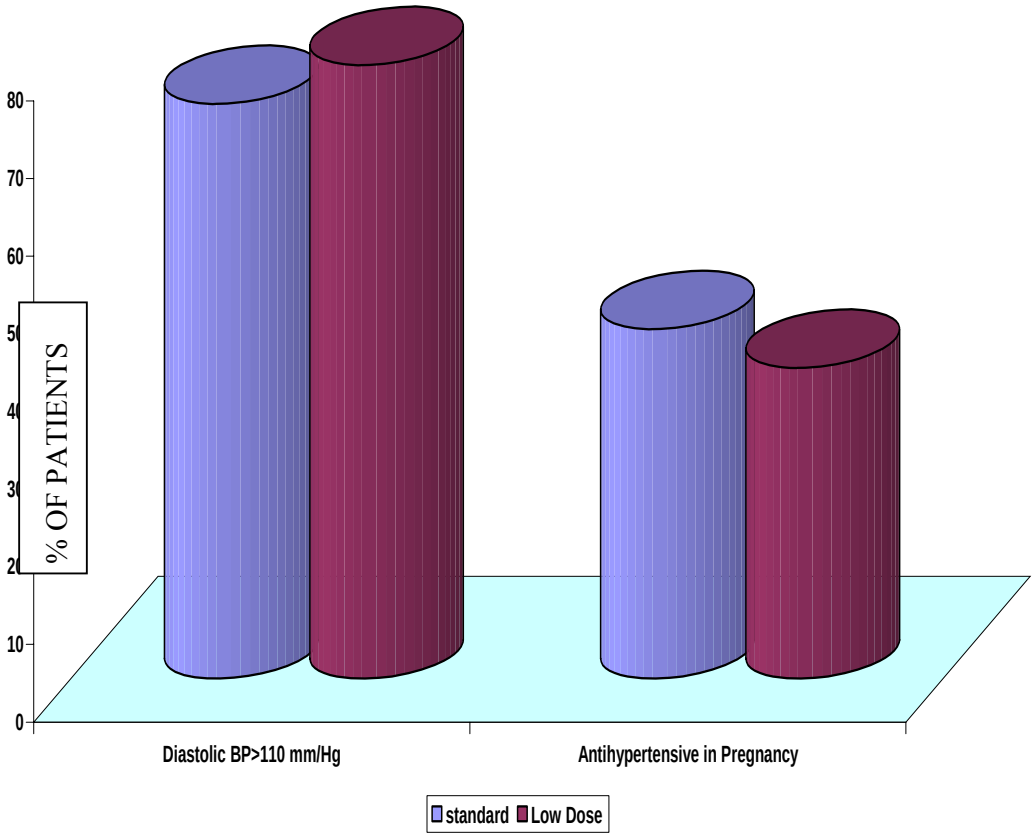
RELIGION



BODY MASS INDEX



BLOOD PRESSURE PARAMETERS



IMMINENT SYMPTOMS

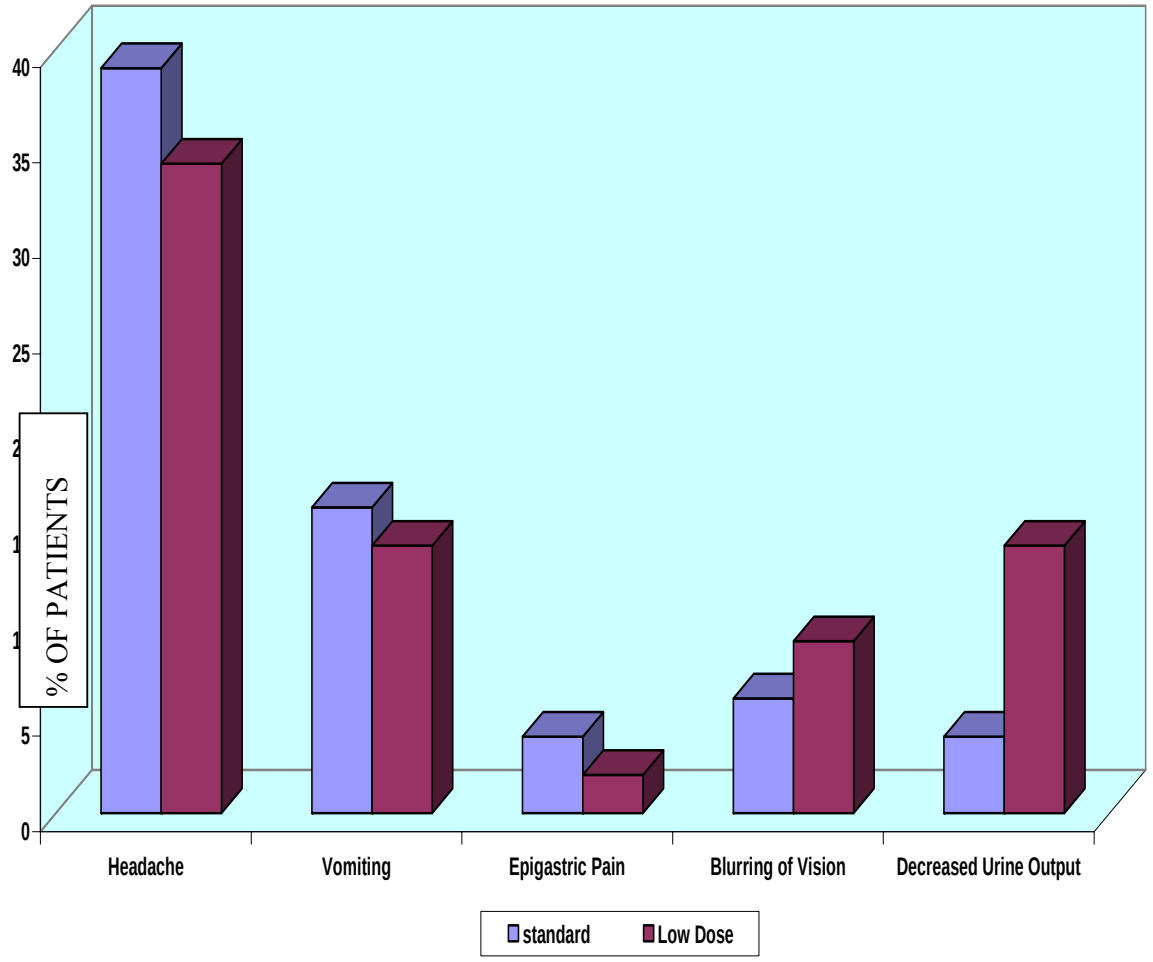
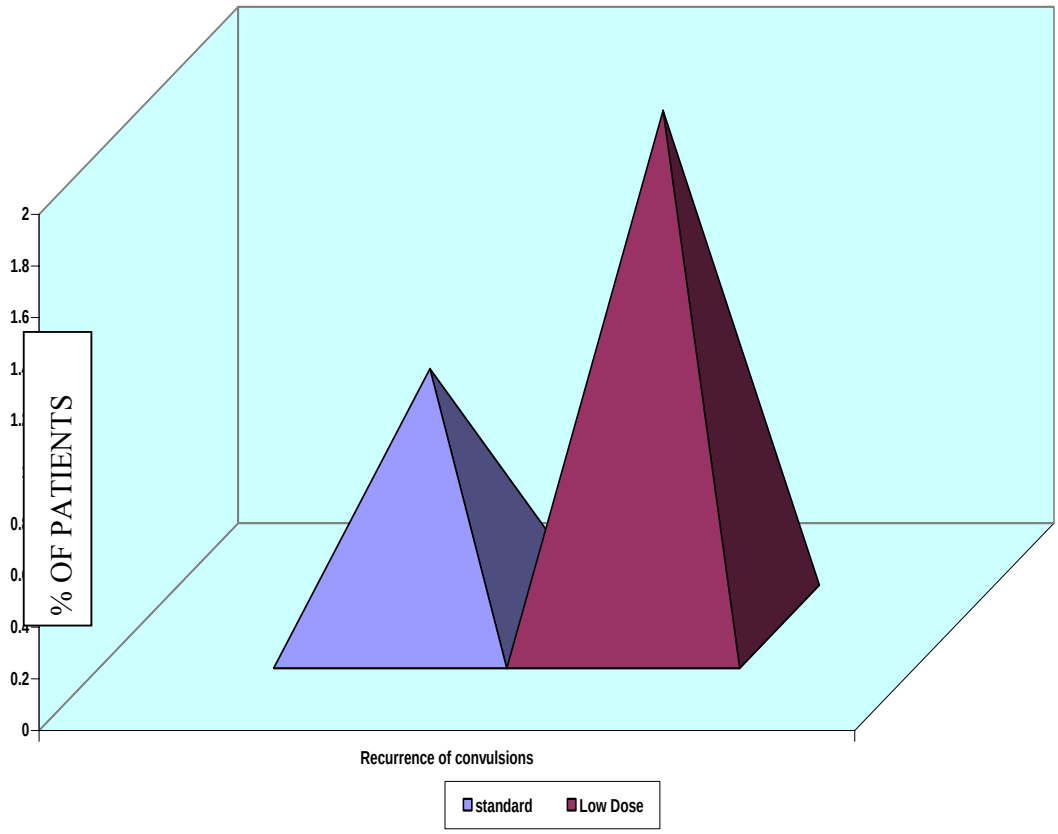


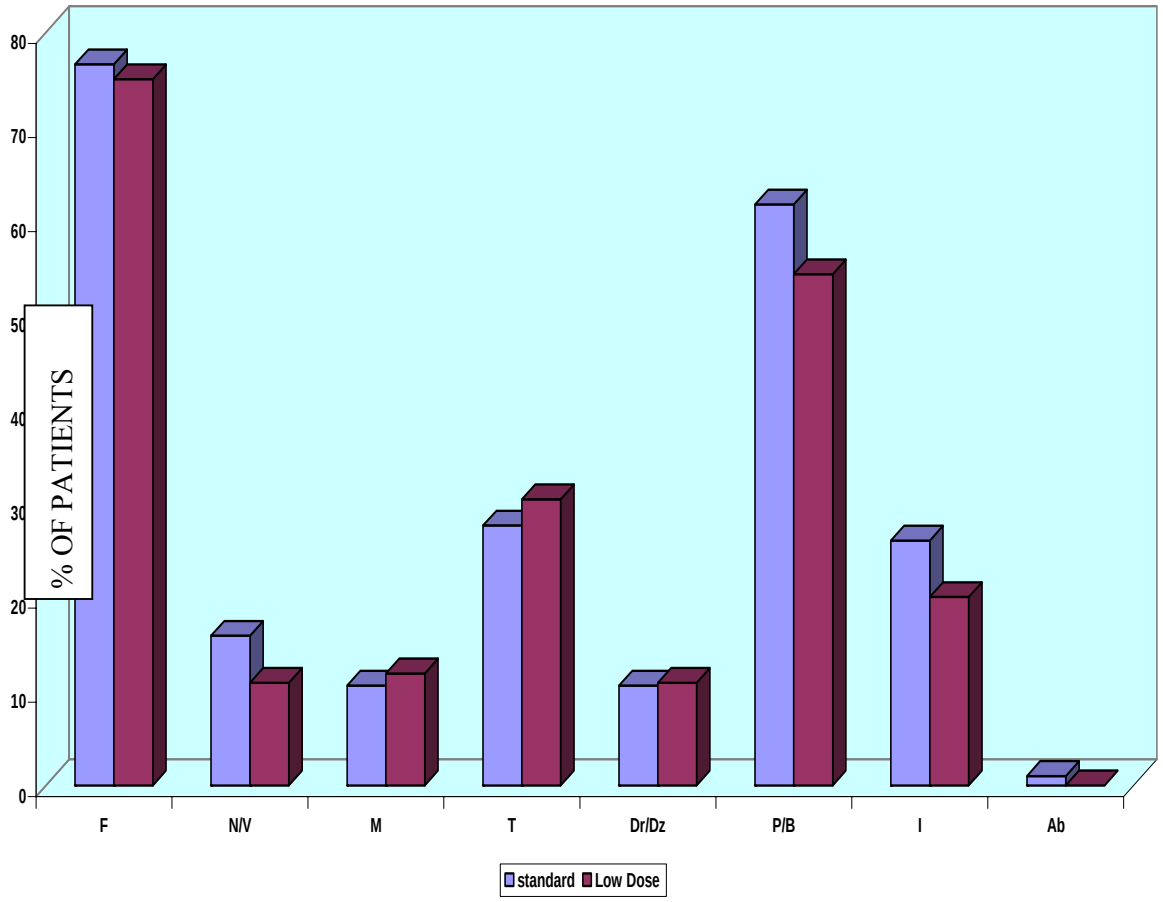
FIGURE - 9

RECURRENCE OF CONVULSIONS



FIGURE– 11

SIDE EFFECTS



F - FLUSHING

N /V – NAUSEA / VOMITING

M – MUSCLE WEAKNESS

T – THIRST

Dr/Dz – DROWSINESS/DIZZINESS

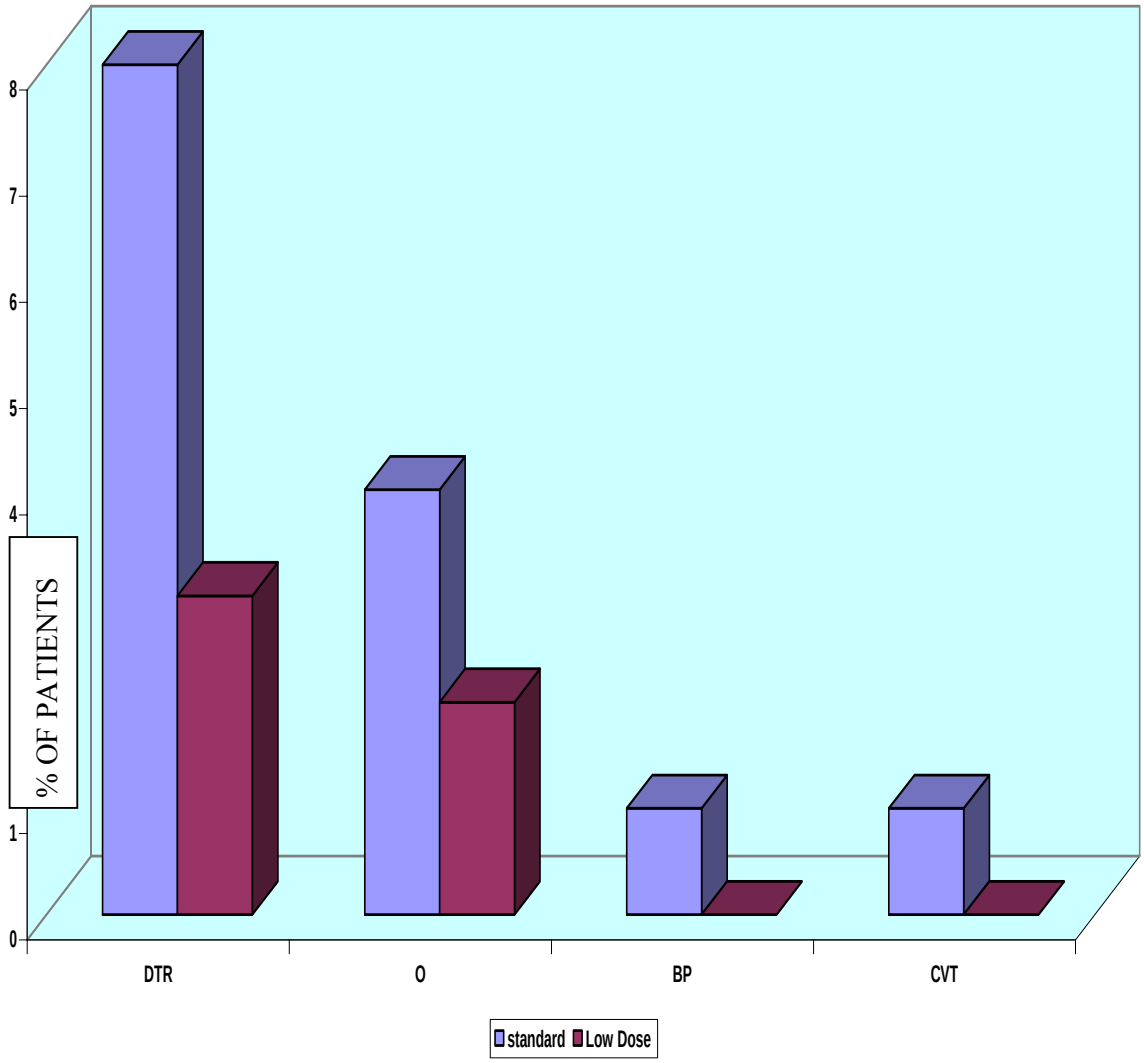
P/B - PAIN/ BURNING

I - INDURATION

Ab - ABSCESS

TABLE – 12

REASON FOR WITHOLDING MAGNESIUM SULFATE



DTR - LOSS OF DEEP TENDON REFLEX

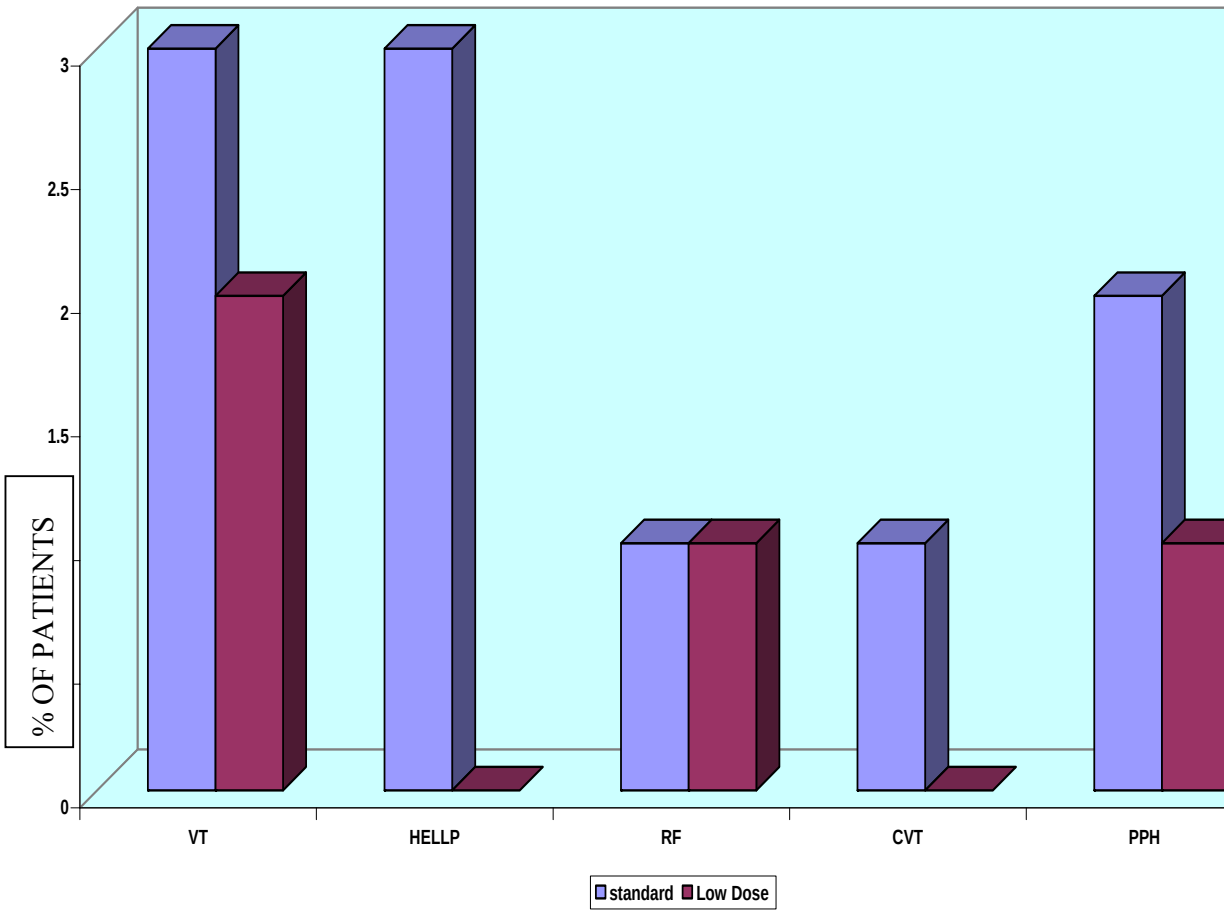
O - OLIGURIA

BP - BLOOD PRESSURE NORMALISED

CVT - CORTICAL VEIN THROMBOSIS

FIGURE -13

COMPLICATIONS



VT - VENTILATOR

RF - RENAL FAILURE

CVT - CORTICAL VEIN THROMBOSIS

PPH - PPH REQUIRING BLOOD TRANSFUSION

FIGURE - 14

MODE OF DELIVERY

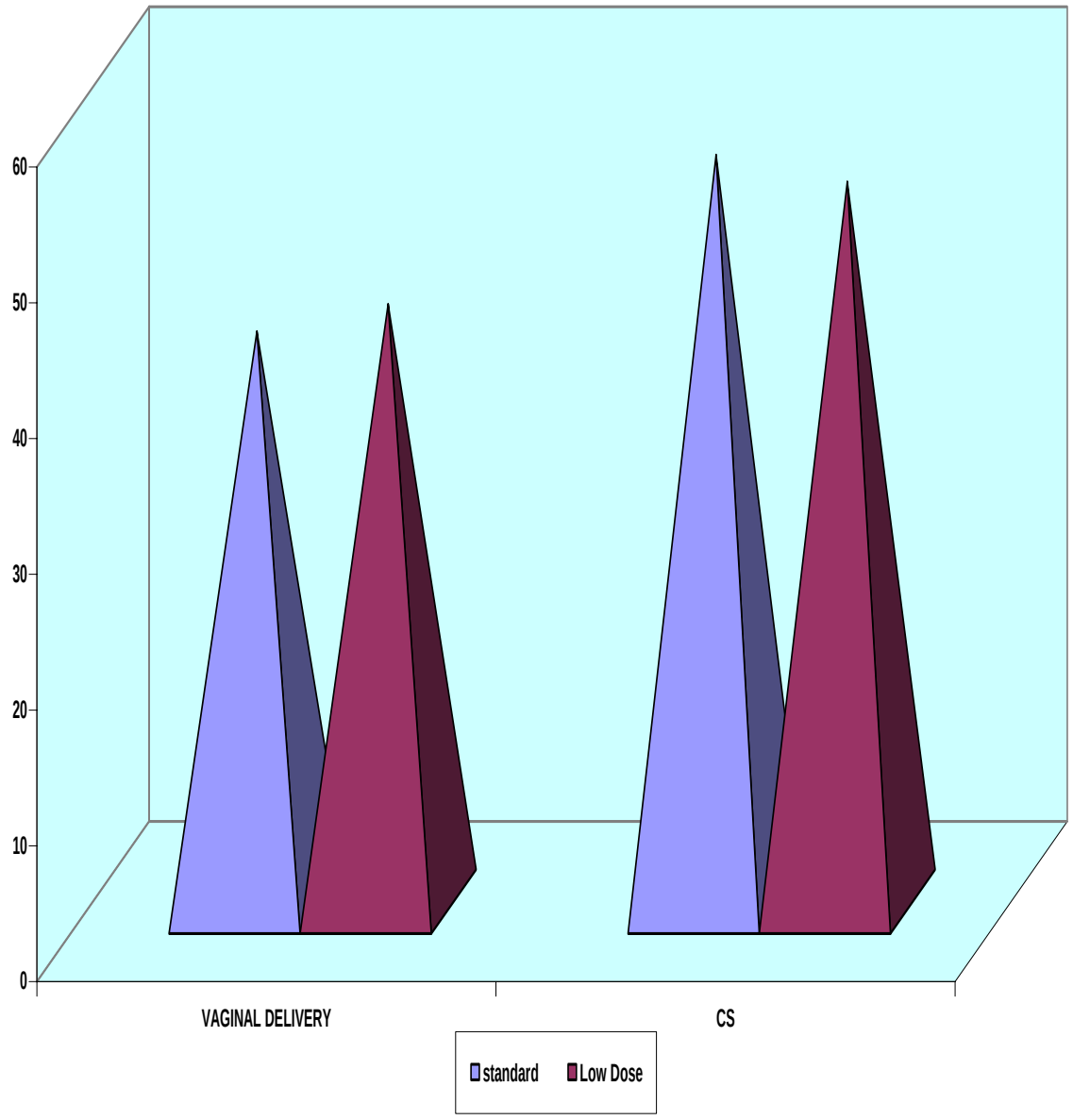
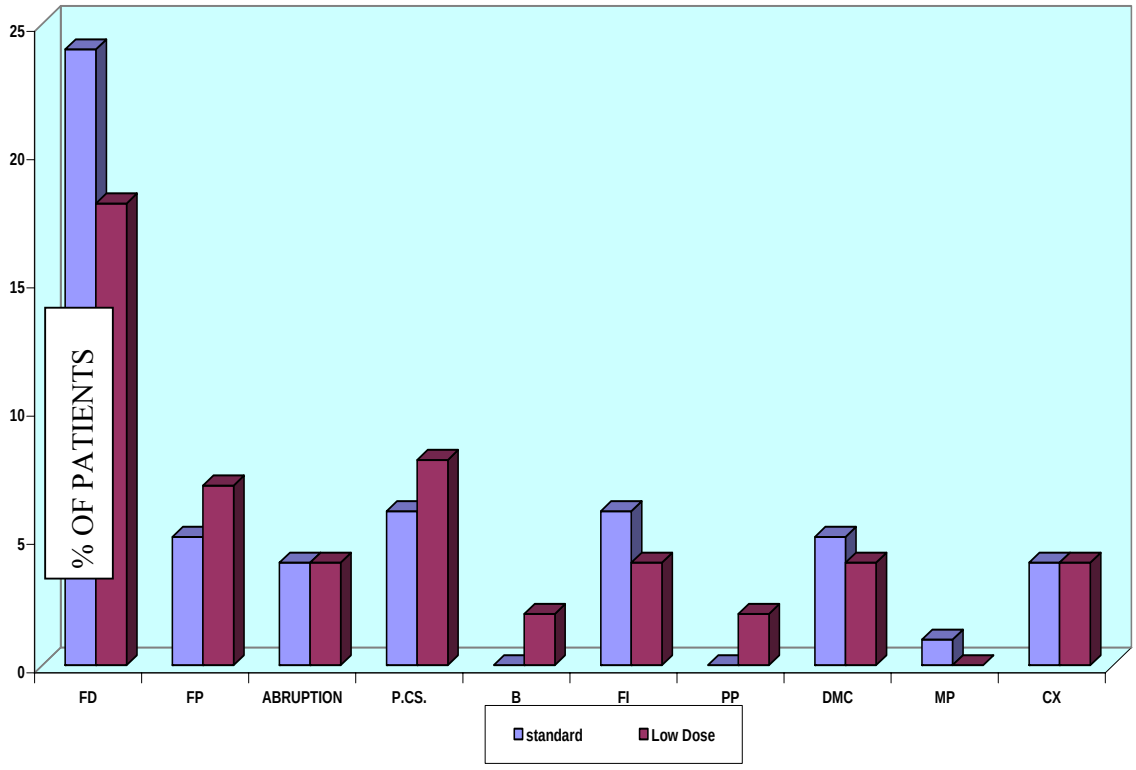


FIGURE - 15

INDICATION FOR CAESAREAN SECTION



FD - **FETAL DISTRES**

FP - **FAILURE TO PROGRESS**

P.CS - **PREVIOUS LSCS**

B - **BREECH**

FI - **FAILED INDUCTION**

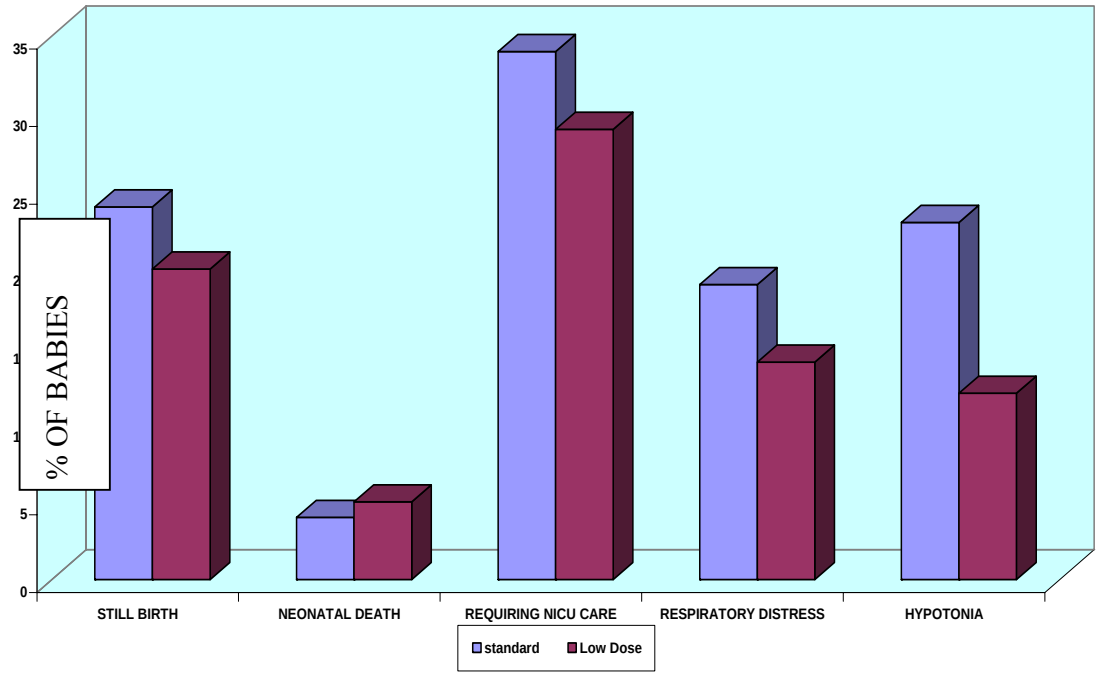
PP - **LOWLYING PLACENTA**

DMC - **DETERIORATING MATERNAL CONDITION**

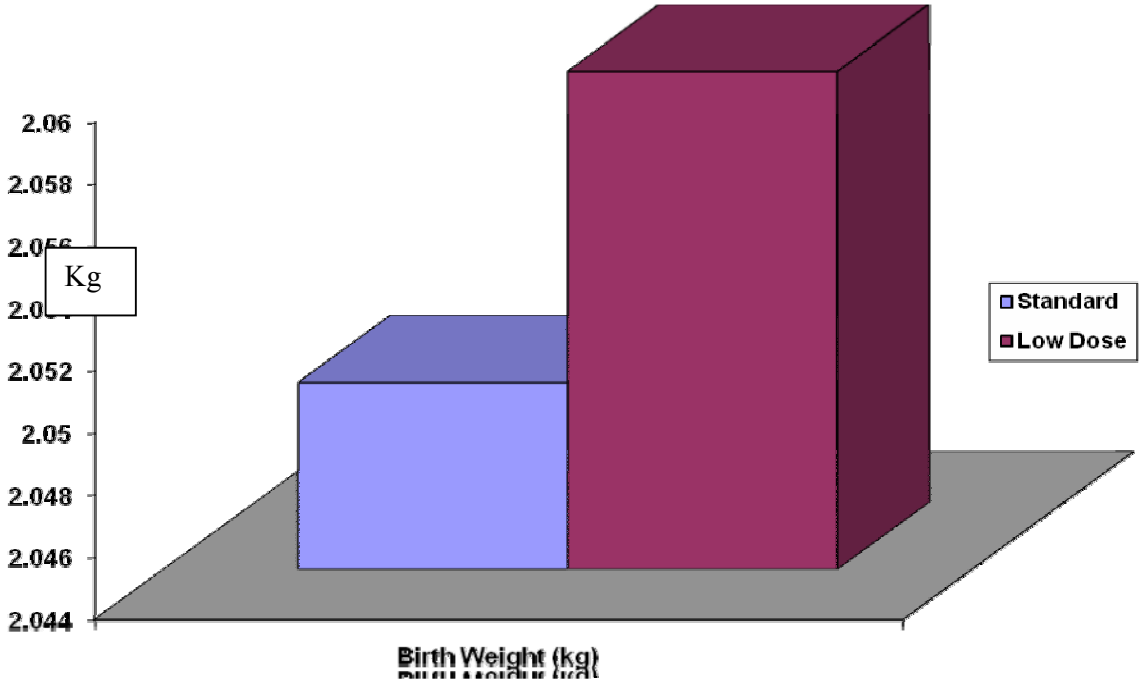
MP - **MULTIPLE PREGNANCY**

CX - **UNFAVOURABLE CERVIX**

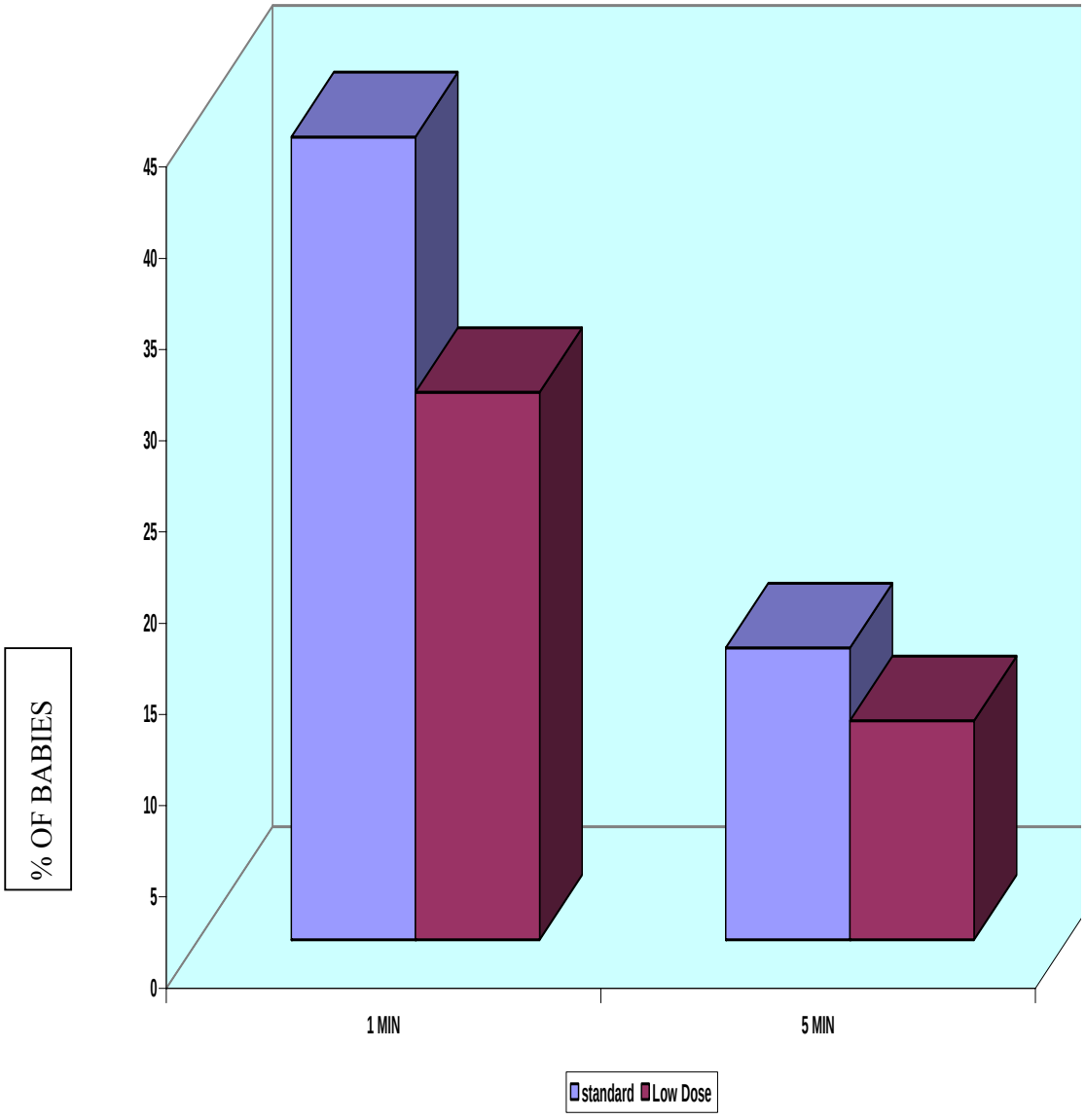
PERINATAL OUTCOME



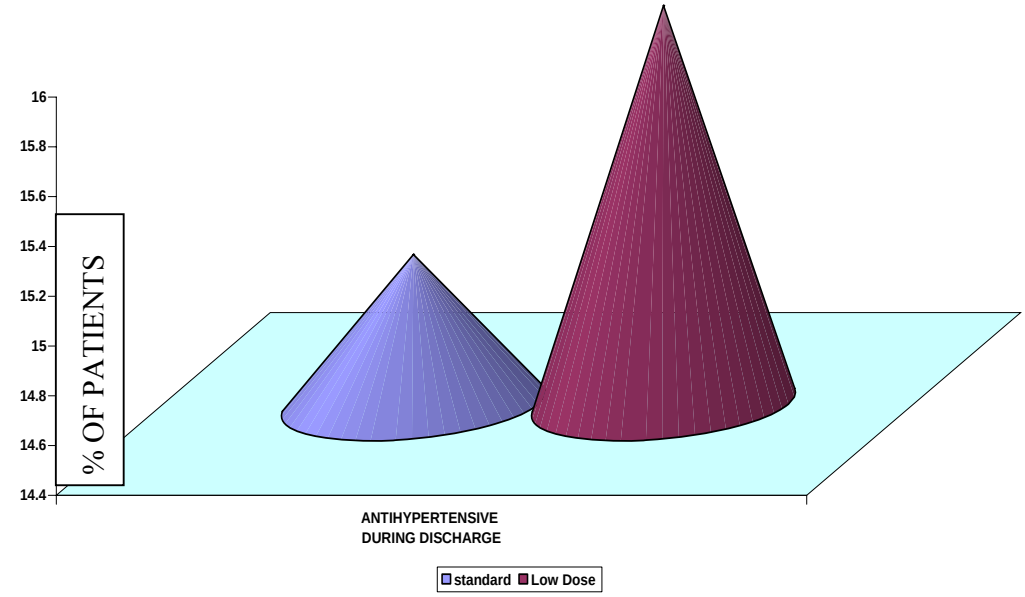
BIRTH WEIGHT

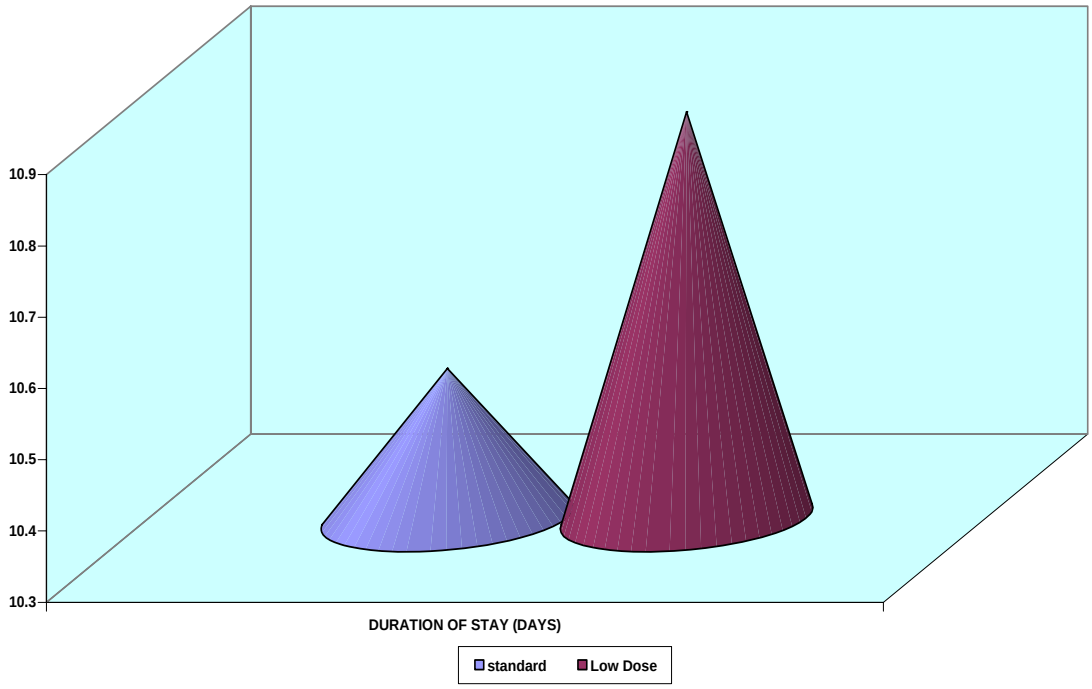


APGAR <7



POSTNATAL DATA


16
15.8
15.6
15.4
15.2
15
14.8
14.6
14.4
% OF PATIENTS
ANTIHYPERTENSIVE
DURING DISCHARGE
standard
Low Dose


10.9
10.8
10.7
10.6
10.5
10.4
10.3
DURATION OF STAY (DAYS)
standard
Low Dose

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
1	Kamatchi	IE	std dose	24	II	HI	BO	19.6	10	PRIMI	40	Y	140/100	3+	H,BV	F,P,V,T				Y	CS	FP	A	2.75	7,8		
2	Fathima	IE	std dose	21	X	M	BO	30.8	6	G2A1	27	Y	160/120	2+		F,M				Y	LN		SB	1.5	-		
3	Maheswari	IE	std dose	24	II	HI	BI	23.4	5	G2P1L1	38	Y	130/100	2+						Y	LN		A	2.4	8,9		
4	Jayalakshmi	IE	std dose	21	XII	HI	B0	20	7	PRIMI	34	Y	170/110	4+	H	F,P,I				Y	LN		SB	1.2	-		
5	Meera	IE	std dose	25	III	HI	BO	22.2	13	G2P1L0	35		140/100	4+	H,BV						CS	FD	A	1.8	4,6	hypotonia	y
6	Jilani	IE	std dose	29	XII	M	BO	40.8	12	G2P1L0	38	Y	150/120	3+	H	F,P,I					CS	Abrubtion	A	3.7	3,6		
7	Pandeswari	IE	std dose	20	II	HI	BO	22.6	12	G3P2L1	30		140/100	1+	E	F,V,P,T					CS	P.CS	A	1.3	6,7	hypotonia	y
8	Sheela	IE	std dose	22	XII	HI	BO	35.13	3	G2P1L1	34	Y	160/90	3+	H,V	F,P,I				Y	LN		A	2..3	7,8		
9	Komala	IE	std dose	32	III	HI	BO	21.3	5	G4P3L3	35	Y	150/100	2+	H	F,T,M					LN		A	1.75	6,7		
10	Varalakshmi	IE	std dose	27	XII	HI	BI		9	G5P4L1	39	Y	170/120	2+	H					Y	LN		A	2.6	5,7		
11	Koteswari	IE	std dose	23	II	HI	BO	19.8	4	PRIMI	37	Y	120/90	4+	H	F,P,I			HELLP	Y	LN		SB	1.5	-		
12	Shakiladevi	IE	std dose	26	XII	M	BO	28.7	9	PRIMI	36		140/90	1+	H,V	F,T					LN		A	2.8	7,8		
13	Shakira	IE	std dose	37	V	M	BO	22.9	6	G3P2L1	32	Y	150/100	2+	H				PPH		LN		SB	1.2			
14	sharmila	IE	std dose	21	X	HI	BI	41.4	19	PRIMI	38	Y	170/110	3+		F,V,D					CS	Abrubtion	SB	1.5			
15	rajeshwari	IE	std dose	20	X	HI	BO	30.2	9	PRIMI	36		150/100	2+	H	F,P,M					CS	FI	A	2.75	7,8		
16	sangeetha	IE	std dose	21	V	HI	UB	24.2	11	PRIMI	38	Y	140/100	4+		F,P,I	↓UO		HELLP	Y	CS	DMC	A	2.1	5,6	Hypotonia, RD	Y
17	devikala	IE	std dose	21	X	HI	BO	22.7	12	PRIMI	40	Y	210/120	3+	V						CS	CX	A	2.4	7.8		
18	amsala	IE	std dose	21	V	HI	BO	25.7	12	PRIMI	38	Y	150/130	3+		F,V,T					CS	FD	A	2.3			
19	satya	IE	std dose	20	VIII	HI	BO	26.3	5	PRIMI	39	Y	160/110	2+		F,P,I,T					LN		A	2.5	7,8		
20	kavitha	IE	std dose	20	V	HI	UB	23.1	6	PRIMI	30		170/110	4+	H,↓UO	F,P,D					LN		SB	1			
21	silambarasi	IE	std dose	23	II	HI	BO	20.8	5	G2P1L1	38	Y	180/110	2+		P,I					LN		A	2.5	6,8		
22	gunasundari	IE	std dose	29	Gr	HI	BO	24.9	12	PRIMI	32		170/120	4+	H,V	F,P					LN		SB	1.4			
23	navanetham	IE	std dose	24	XII	HI	BO	22.8	10	PRIMI	40	Y	150/110	4+	H	P	↓DTR				CS	FD	A	2.5	7,8		
24	famidha	IE	std dose	19	V	M	UB		5	PRIMI	28		170/130	2+	H	F					LN		SB	1			
25	latha	IE	std dose	28	X	HI	BO	23.8	10	G6P2L1A3	37	Y	150/120	4+		F,P					CS	P.CS	A	2.5	7,8		
26	parameshwari	IE	std dose	23	V	HI	UB	22.2	13	PRIMI	32		190/110	3+	H	F,V,P,I,T					CS	Abrubtion	A	1.75	5,6	Hypotonia,RD	Y
27	radha	IE	std dose	20	XII	HI	UB		9	PRIMI	36	Y	180/120	4+		F,P,D					CS	FD	A	2.9	7,8		

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
28	ramya	IE	std dose	22	Gr	HI	UB	41.3	8	PRIMI	30		170/140	4+		F,P					CS	FI	A	1.5	5,6	Hypotonia,RD	Y
29	lakshmi	IE	std dose	23	X	HI	UB		9	G2A1	30		180/120	4+	↓V,BV,↓UO						CS	DMC	E	1	6,7	Hypotonia,RD	Y
30	meena	IE	std dose	23	X	HI	BO	29	14	G2P1L1	40		180/130	2+	H	F,M					CS	FP	A	1.6	7,8	hypotonia RD	Y
31	jeeva	IE	std dose	20	V	HI	UB	21.2	9	PRIMI	41		140/100	1+	H,V	F,P,I					CS	FD	A	2.9	7,8	RD	Y
32	anandhi	IE	std dose	30	VIII	HI	BO	38.9	11	PRIMI	22	Y	180/140	3		F,P,I			MRP		LN		SB	0.6			
33	vidyalatha	IE	std dose	33	V	HI	BI	18	22	G2P1L1	35	Y	200/100	1	V	F,T				Y	CS	P.CS	A	1.25	6,7	IUGR	Y
34	gomathy	IE	std dose	22	II	HI	BO	27.88	12	PRIMI	40		150/110	3		F,V,P,I					LN		A	2.8	7,8		
35	thayar	IE	std dose	22	II	HI	BO		22	PRIMI	30		150/110	2	H,BV						CS	FI	SB	0.5			
36	thilagam	IE	std dose	24	XII	HI	BO	23.4	11	G2P1L1	40	Y	180/130	2		F,D					LN		A	1.75	6,8	hypotonia	y
37	sujatha	IE	std dose	28	II	HI	BO	27.6	29	G2P1L1	33		180/130	3		F,V,P,T					CS	FI	A	2.1	6,8		y
38	vijaylakshmi	IE	std dose	23	XII	HI	BO	24.3	8	PRIMI	40		150/110	1	H,E						CS	FD	A	3.2	7,8		
39	shameena	IE	std dose	19	X	M	UB	38.7	11	G3P1L1A1	35	Y	150/110	1	H	F,V,P	↓UO				CS	P.CS	A	2.8	7,8		
40	vasantha	IE	std dose	21	V	HI	BO	23.4	5	PRIMI	40	Y	180/130	2		F,M,T					LN		A	2.25	7,8		
41	valarmathy	IE	std dose	19	XII	HI	BO	26.7	12	PRIMI	31		140/100	4	H						LN		A	1.25	6,7	hypotonia,RD	Y
42	vijaya	IE	std dose	35	III	HI	BI	29.5	8	G2P1L1	36		170/110	2		F,P,I,T	↓DTR				CS	P.CS	A	2.25	7,8		Y
43	deivanjali	IE	std dose	26	XII	HI	BO	23.3	9	G3P2L2	40		140/110	4	H,V,E	F,P,I					LN		A	1.6	7,8		
44	poongodi	IE	std dose	26	X	HI	BO	30.8	10	G2P1L0	37		170/114	3							CS	FD	A	3	3,7	RD,Hypotonia	Y
45	jyothilakshmi	IE	std dose	21	II	HI	BO	21.7	9	PRIMI	40		180/120	3	H	F,P,I					CS	FD	A	2.1	6,8	RD	Y
46	kavitha	IE	std dose	21	UE	HI	BO	26.2	7	G3P1L1A1	40	Y	170/110	3		F,P,T			PPH		CS	Abrubtion	A	3	5,6	hypotonia	Y
47	Shaira	IE	std dose	23	V	M	BI	26.2	5	PRIMI	38	Y	160/110	3			BP				LN		SB	1.6	-		
48	devaki	IE	std dose	31	VIII	HI	BI	27.08	8	PRIMI	40		170/110	3							CS	FD	A	3.4	6,8		
49	manimegalai	IE	std dose	27	III	HI	BO	25.8	19	G3P2L2	35		150/110	3		F,P					LN		A	1.5	6,7	hypotonia	y
50	saroja	IE	std dose	20	V	HI	BO	28	12	PRIMI	36	Y	150/110	2		F,T					CS	FD	A	2.2	7,8		y
51	jyothi	IE	std dose	32	X	HI	BO		20	G2PL1	36		170/110	3		F,P,M					LN		A	2.7	7,8		
52	subhashini	IE	std dose	24	UE	HI	UB	27.6	10	G2P1L1	40		140/110	3		F,P,V					CS	FD	A	2.1	6,8	hypotnia	y
53	kala	IE	std dose	23	VIII	HI	BO	20.3	8	PRIMI	40	Y	160/120	3		F,P,V					CS	FD	A	2.2	8,9	RD	y
54	aprose	IE	std dose	35	X	C	BO		22	G4P1L0A2	33		180/120	4							CS	FD	A	1.25	4,7	RD,hypotonia	y

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
55	sasikala	IE	std dose	29	XII	HI	BO	25.9	8	PRIMI	38	Y	160/110	2		F,P,D	↓DTR				CS	DMC	A	3.25	6,8		
56	ezhilarasi	IE	std dose	24	VIII	HI	BI	23.2	13	G2P1L1	38		150/110	3							CS	FD	A	1.9	7,8	hypotonia,RD	y
57	nadhiya	IE	std dose	24	XII	HI	BI	27.1	9	PRIMI	40	Y	170/120	3		F,P					CS	FD	A	2.6	6,7		
58	sumathi	IE	std dose	38	III	HI	BI	25.9	9	G2A1	38	Y	190/110	3		F,P,V					CS	FD	A	2.7	6,8		
59	swaroopa	IE	std dose	21	V	HI	B0	26.8	2	PRIMI	36		130/120	2	H,V	F,P,I,A					LN		E	2.6	3,6	RD,hypotonia	y
60	poongodi	IE	std dose	24	XII	HI	BO	29.4	7	PRIMI	37		140/110	3							CS	CX	A	3.5	7,8		
61	ramani	IE	std dose	24	X	HI	BO	30	8	PRIMI	37	Y	170/110	1	H,V						CS	FD	A	2.75	7,8		
62	prabhavathy	IE	std dose	23	X	HI	BO	22.9	8	PRIMI	37	Y	190/130	4	H,V,BV	F.P,I	↓DTR				CS	DMC	A	2.4	3,7	RD,hypoyonia	y
63	sumathi	IE	std dose	21	VIII	HI	BO		10	PRIMI	29		180/130	3	H,E	F,V,P					LN		SB	0.75			
64	kanaga	IE	std dose	24	UE	HI	UB	26.7	8	PRIMI	37		160/110	4		F,P,D					CS	FD	A	2.3	8,9		
65	bhuvaneswari	IE	std dose	25	X	HI	UB		5	PRIMI	29		150/110	4		F,P,I				Y	LN		SB	0.6			
66	karpagavalli	IE	std dose	25	V	HI	BO	20.2	18	PRIMI	36		170/120	2		F,M	↓UO		RF		CS	FI	E	1.2	4,7	hypotonia,RD	Y
67	vasanthi	IE	std dose	22	VIII	HI	BO	25.5	13	PRIMI	39	Y	150/100	3	H,V						LN		A	2.7	6,7		
68	bharathi	IE	std dose	25	III	HI	BO	30	3	G2P1L1	34		150/110	2	H	F,P,I				Y	LN		A	2.2	6,7		
69	satya	IE	std dose	27	Gr	HI	UB		9	PRIMI	34		210/140	3	H,V,BV	F,T				Y	CS	FD	A	2	6,7		
70	ezhilarasi	IE	std dose	20	II	HI	BO	26.9	15	G2A1	34		150/110	4				HELLP,VENT			CS	DMC	A	2	3,5	RD,Hypotonia	Y
71	sudha	IE	std dose	18	X	HI	UB		13	PRIMI	34		140/110	3	H,V	F,V,P	↓DTR				CS	P.CS	A	3.25	6,8		
72	uma	IE	std dose	28	X	HI	BO	34.2	7	G2P1L1	38	Y	150/110	3	H	F,V					CS	FP	A	3.25	6,7		
73	alamelu	IE	std dose	22	Gr	HI	BO	22.8	8	PRIMI	40		160/110	2	H	P					LN		A	1.75	6,8		y
74	malarkodi	IE	std dose	27	XII	HI	BO	23.7	16	G2P1L1	36		160/110	3		F,P,D	↓DTR				LN		A	1.8	6,8		
75	shyamaladevi	IE	std dose	25	V	HI	BO		9	PRIMI	37		150/110	2	H	F,T					CS	FD	A	2.75	6,7		
76	nisha	IE	std dose	22	X	HI	BO	26.8	8	PRIMI	36		150/110	1	H,V	F,P,I					CS	FD	A	2.6	6,8		
77	lakshmi	IE	std dose	25	VIII	HI	BO	21	8	PRIMI	39	Y	150/120	3	V	F,P,M				Y	CS	FD	A	2.4	6,8	IUGR	Y
78	padma	IE	std dose	22	X	HI	BO		17	PRIMI	28		160/110	1	H,V,BV	F,P,I					LN		SB	1	,		
79	vijajlaksnmi	IE	std dose	23	VIII	HI	UB	25.7	16	G3P1L1A1	39		170/120	4	H,↓UO	F,P,T	↓DTR	ONVULSION			LN		SB	1.5			
80	usha	IE	std dose	27	Gr	HI	BO	28	8	PRIMI	40	Y	130/100	2	H						CS	CX	A	2	5,6	RD	Y
81	jayaprada	AP	std dose	20	Gr	HI	BO	25.6	19	PRIMI	37		170/120	4		F,P,V,T					LN		E	2.3	3,5	RD,Hypotonia	Y

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
82	selvi	AP	std dose	23	II	HI	UB	18.4	11	G2P1L0	28		130/104	3		-	CONVULSIONS		VENTILATOR		CS	FP	A	2	4,6	Hypotonia,RD	Y
83	durgadevi	AP	std dose	21	UE	HI	UB	18.4	14	PRIMI	28		140/100	4		F,P,D					LN		SB	1.5			
84	dhatchayani	AP	std dose	20	X	HI	BO	22.3	10	G2P1L1	36		140/90	3		F,P,T	↓UO				CS	CX	A	2	5,6	Hypotonia	Y
85	devi	AP	std dose	24	VIII	HI	BO	24.2	22	PRIMI	37		190/130	2	H,E	F,P,M,I					CS	TWINS	A	2.1./1.7	4,6	RD,Hypotonia	Y
86	babitha	AP	std dose	22	XII	HI	BO	24.8	9	G2P1L1	39	Y	180/120	4	H	F,P,T					CS	FP	A	2.75	7,8		
87	kanikakumari	AP	std dose	24	Gr	HI	BO	21.9	11	PRIMI	28		210/130	4	H,V						LN		SB	2.4			
88	vimala	AP	std dose	19	XII	HI	BO	27.9	11	PRIMI	38	Y	170/110	4					VENTILATOR		CS	FD	A	3.35	7,8		
89	chandra	AP	std dose	21	X	HI	BO	19.2	9	PRIMI	38	Y	150/110	3		F,P,D,I					CS	FD	A	3.5	7,8		
90	nandhini	AP	std dose	20	X	HI	BO	19.2	5	PRIMI	27	Y	230/90	4		F,P,M				Y	LN		SB	0.7			
91	sasikala	AP	std dose	23	VIII	HI	UB	27.4	10	PRIMI	31		170/100	3		F,P					LN		SB	1.3			
92	meena	AP	std dose	18	Gr	H	UB	25	6	G2P1L1	28		150/100	3		F,P,D,I					LN		SB	1			
93	janet	AP	std dose	39	VIII	HI	BO	23	8	G3P2L2	28	Y	150/100	2		F,P,I				Y	LN		SB	1.6			
94	Meera	AP	std dose	19	Gr	HI	B0	28.4	12	PRIMI	35	Y	160/110	3		F,P,I					LN		SB	1.6			
95	manimegalai	AP	std dose	22	UE	HI	B0	26.6	11	PRIMI	33		160/110	4		F,T	↓DTR				CS	FI	A	1.75	6,7		Y
96	banu	AP	std dose	21	XII	HI	UB		5	PRIMI	33		190/130	3							LN		SB	0.45			
97	reeta	AP	std dose	20	II	C	BO	26	11	PRIMI	22		200/120	4							LN		SB	0.45			
98	lakshmi	PP	std dose	22	UE	HI	BO		12	P1L1		Y	170/110	2		F,P,I					LN		A	1.8	6,7		
99	navanetham	PP	std dose	24	VIII	HI	BO		11	P1L1			160/100	3		F,P					CS		A	2.5	7,8		
100	saranya	PP	std dose	21	XII	HI	BO		20	P2L2		Y	150/100	2			CVT		CVT		CS		A	1.8	5,6		

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
1	sumathi	IE	low dose	20	V	HI	BO	24.6	9	PRIMI	32		140/110	3	H,BV	F					CS	Abruption	A	1.2	6,7	hyopotonia	Y
2	asha	IE	low dose	21	X	HI	BO	33.8	6	PRIMI	35	Y	140/110	2	H	P,M,I					LN		E	2	3,6		Y
3	punithavalli	IE	low dose	30	II	HI	BI	29.2	10	G2P1L1	38	Y	180/110	2	H	F,V,P,I					CS	P.CS	A	2.75	7,8		
4	reeta	IE	low dose	27	V	HI	BO	21	6	PRIMI	26		160/110	2	H,↓UO,E	F,T					LN		SB	0.9			
5	valarmathy	IE	low dose	22	II	HI	BO	40.4	11	PRIMI	39	Y	200/120	2	H,V	F,D,P,T	↓DTR			Y	CS	FD	A	2.9	8,9		
6	anushalini	IE	low dose	24	VIII	HI	OB	30	10	PRIMI	37	Y	130/100	2							LN		A	2	7,8		
7	khadeeeja	IE	low dose	22	V	M	B	25.3	7	PRIMI	33		150/120	4		F,P,I				Y	LN		SB	1			
8	shalini	IE	low dose	20	XII	HI	BO	24.8	10	PRIMI	38	Y	130/90	3		F,V,T				Y	CS	FD	A	3.1	7,8		
9	ganasekhari	IE	low dose	27	VIII	HI	BO	27.8	8	PRIMI	34		170/110	2	H	F,P,I					CS	FI	A	2.85	7,8		
10	malliga	IE	low dose	28	V	HI	BI	31.5	8	PRIMI	36	Y	140/110	2	BV						CS	B	A	2.7	7,8		
11	anitha	IE	low dose	19	X	HI	UB	24.2	9	PRIMI	40		140/90	1	H,V,BV,E	P					CS	FD	A	3.2	7,8		
12	muthulakshmi	IE	low dose	24	V	HI	BO	29.2	20	PRIMI	32		140/100	1	H	F,D,P,I					LN		SB	1.5			
13	vijaya	IE	low dose	25	X	HI	BO	20.8	3	G2P1L1	38	Y	170/120	4							LN		SB	1.5			
14	revathy	IE	low dose	22	VIII	HI	BO	20.9	8	PRIMI	28		150/110	3	↓UO	F,M					LN		SB	1.1			
15	jayapoorna	IE	low dose	29	V	HI	BO	34.7	11	PRIMI	28		160/100	4		F,D,T					CS	FI	SB	1.2			
16	yamini	IE	low dose	25	II	HI	BI	29.8	20	G2P1L1	35		140/100	2	H	F,P					CS	P.CS	A	3.1	7,8		
17	maheswari	IE	low dose	22	VIII	HI	BI	26.1	9	PRIMI	39	Y	140/100	2	H,V	F,P,I,T					CS	FD	A	2.9	7,8	hyopotonia,RD	Y
18	usha	IE	low dose	20	X	HI	BO	21.6	10	PRIMI	36		160/110	4							CS	Ab	A	1.7	4,6		Y
19	sulekha	IE	low dose	21	XII	HI	BO	37.3	11	PRIMI	34		170/120	2		F,M,P				Y	CS	FD	A	3	7,8		
20	kiruba	IE	low dose	28	V	HI	BI	37.5	16	G2P1L1	26		130/100	4		F,V,D				Y	CS	FP	A	2.25	,78		
21	pavithra	IE	low dose	20	X	HI	BI	26.2	9	PRIMI	37	Y	150/90	2							LN		A	2.7	7,8		
22	kavitha	IE	low dose	22	XII	HI	BO		7	G3A2	33		170/100	3	H,↓UO	F,P,T					LN		SB	0.5			
23	madhavi	IE	low dose	28	VIII	HI	BI	32.4	22	G2P1L1	37	Y	160/100	4	↓UO						CS	FI	A	2.8	7,8		
24	sasikala	IE	low dose	20	Gr	HI	BI	23.9	8	G3P2LI	37		170/110	3	H,BV	F,P,I					CS	P.CS	A	2.75	7,8		
25	jayanthi	IE	low dose	29	XII	HI	BO		7	PRIMI	38	Y	150/110	3	H,V	F,P					CS	FD	A	1.6	6,8	IUGR	Y
26	sangeetha	IE	low dose	18	VIII	HI	BO	20.9	8	PRIMI	32		180/120	4	H,↓UO	F,P,T					LN		A	1.75	7,8		
27	jayanthi	IE	low dose	30	XII	HI	BI	21.5	8	PRIMI	32		140/100	1	H,V	F					LN		A	1.5	7,8	hypotonia	Y
28	khadarbee	IE	low dose	20	V	HI	BO	23.9	7	PRIMI	39	Y	140/110	3	V	F,P					CS	FP	A	3.1	7,8		
29	bhuvaneshwari	IE	low dose	23	XII	HI	BO	39	8	G2A1	32	Y	150/110	3		F					LN		A	2.5	6,7		
30	vijaya	IE	low dose	28	UE	HI	UB	24.7	12	PRIMI	24		130/94	3	↓UO	F,V,M					LN		A	1.5	7,8		

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
31	thenmozhi	IE	low dose	28	X	HI	BO	24.7	18	G2P1L1	33		180/120	4	H,↓UO					Y	CS	PP	A	1.2	7,8	RD	Y
32	bharathy	IE	low dose	23	XII	HI	BO	22.4	21	G2P1L1	35		150/110	4		F,P,I					LN		A	1.6	7,8	RD	Y
33	subhashini	IE	low dose	23	V	HI	BI	24.4	12	G4P2L2A1	36	Y	180/130	2	↓UO	F,D,P					CS	FD	A	1.6	5,6	RD	Y
34	elavarasi	IE	low dose	23	II	HI	BO	26.9	12	PRIMI	32		130/110	4	H,V	F,P,T					LN		SB	0.9			
35	chithra	IE	low dose	30	X	HI	BO	34.6	5	G3P1L1A1	37	Y	130/110	3	H	F					LN		A	2.9	7,8		
36	sumathi	IE	low dose	30	V	HI	BO	30.8	12	G2P1L0	30		160/100	3		F,V,D					LN		E	1.4	3,5	hypotonia RD	Y
37	usha	IE	low dose	20	X	HI	BO	21.6	21	PRIMI	36		160/110	4		F,P,T					CS	Abruption	A	1.7	3,4	hypotonia	Y
38	srividhya	IE	low dose	31	X	HI	BO		4	G2A1	30		150/110	3	H,V	F,P,I					LN		SB	0.9			
39	jennifer	IE	low dose	20	VIII	C	BO	24.8	12	PRIMI	38	Y	130/90	1	H,V						CS	FI	A	1.8	7,8		
40	latha	IE	low dose	25	XII	HI	BO	26	17	PRIMI	36		140/110	2	H	F,V,P					CS	FD	A	2.75	3,6	hypotonia	Y
41	shanthi	IE	low dose	24	II	HI	BO	25.8	5	PRIMI	37	Y	180/150	3	H	F,P,T					LN	FD	E	1.5	6,7	IUGR	Y
42	lakshmi	IE	low dose	19	V	HI	BO	27.6	17	PRIMI	36		200/110	1	H	F,P,T					CS	B	A	2.25	7,8	IUGR	
43	pushpa	IE	low dose	26	XII	HI	BO	38.5	8	G2P1L0	37	Y	170/110	3	H,↓UO	F,V,M	↓DTR				CS	FD	A	2.4	6,7	RD	Y
44	fathima	IE	low dose	23	II	M	BO	25.5	16	G2P1L1	36		170/120	2	H						CS	CX	A	1.7	7,8		Y
45	kavitha	IE	low dose	23	V	HI	BO	28.6	8	PRIMI	40		150/110	2							CS	FD	A	3.4	5,8		
46	anitha	IE	low dose	25	Gr	HI	BI	20.9	9	PRIMI	37	Y	140/120	3	H	F,P,I					CS	FD	A	2	7,8	RD	Y
47	muniamma	IE	low dose	30	UE	HI	UB	24.4	11	G4P2L2A1	34	Y	170/120	3	H	F,P,M					CS	PP	A	1.6	6,7	RD	
48	sangeetha	IE	low dose	23	XII	HI	BO	22.7	5	PRIMI	38		180/120	3							LN		SB	0.9			
49	vijaya	IE	low dose	23	V	HI	BO	25	4	G3P1L1A1	34		170/110	3		F,P,I					LN		A	1.6	7,8		Y
50	kavitha	IE	low dose	24	XII	HI	BI	31.5	6	PRIMI	33	Y	150/110	2		F,P,T					LN		SB	0.9			
51	amudha	IE	low dose	26	II	H	BO	19.5	6	G2P1L1	30		180/130	2							LN		SB	1.3			
52	naveena	IE	low dose	33	X	HI	BI	38.8	9	G3P1L1A1	37	Y	150/110	3	H,V,BV	F,M					CS	FD	A	2.3	7,8	RD	Y
53	pavithra	IE	low dose	21	XII	HI	BI	22.5	9	PRIMI	37	Y	160/120	1	H				PPH		LN		SB	1			
54	jayalakshmi	IE	low dose	22	VIII	HI	BO	22.9	20	G2P1L0	36		140/110	2	H,V	F,P,I	CONVULSION				CS	DMC	A	1.8	5,7		Y
55	vanitha	IE	low dose	27	UE	C	UB		22	G2P1L1	35	Y	160/110	3		F,P					CS	P.CS	A	2	6,7		Y
56	sobana	IE	low dose	27	XII	HI	BO	29.2	9	G3P1L1A1	36		170/120	3	↓UO		↓UO				CS	DMC	A	2.25	7,8		
57	dharmeth	IE	low dose	36	X	M	BO	40.8	18	G2P1L1	37	Y	200/110	4	H	F,P,I					CS	P.CS	A	2.5	7,8		
58	kuntha	IE	low dose	20	XII	HI	BO	29.7	9	PRIMI	38	Y	150/110	2	H,↓UO						CS	FP	A	2.4	7,8		
59	sumathi	IE	low dose	27	UE	HI	UB		18	PRIMI	36		160/110	3	H,V	F,D,T					LN		A	1.6	7,8		
60	shanthi	IE	low dose	24	Gr	HI	BO		10	PRIMI	37		160/120	2	H,V	F,P,I					CS	CX	A	2.5	7,8		

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
61	bharathy	IE	low dose	21	V	HI	UB		24	PRIMI	37		150/110	2							CS	CX	A	1.5	3,5	hypotonia	Y
62	mayuri	IE	low dose	23	XII	HI	BO		8	PRIMI	38	Y	170/120	4		F,P,M					CS	FD	A	3.1	7,8		
63	asmath	IE	low dose	21	XII	M	UB		8	G3P2L2	39		160/110	3							LN		A	1.75	4,6	Hypotonia	Y
64	nalini	IE	low dose	22	V	HI	BO	19	13	PRIMI	34		150/100	2							LN		A	2.9	7,8		
65	selvi	IE	low dose	21	V	HI	BO	20	10	G2A1	37	Y	130/100	3		F,P,T	↓DTR				LN		A	2.75	7,8		
66	jaya	IE	low dose	22	VIII	HI	BO	23.8	11	PRIMI	39	Y	160/120	4		F,V,P					CS	FD	A	3.25	7,8		
67	usha	IE	low dose	27	II	HI	UB	24.5	10	G2P1L1	37		180/110	3	H	F,P					CS	FD	A	2.6	7,8		
68	vijaylakshmi	IE	low dose	20	V	HI	BO	26.7	14	PRIMI	33		160/106	2		F,D,P				Y	LN		A	.25,1.2	5,6	RD	Y
69	farhana	IE	low dose	25	X	M	UB		8	PRIMI	34		160/110	3							LN		E	1.25	3,5		
70	mala	IE	low dose	24	XII	HI	BO	22.8	5	PRIMI	40	Y	160/110	3		F,P,I				Y	LN		A	1.25	5,,6	RD,hypotonia	Y
71	vanitha	IE	low dose	22	Gr	HI	BO	17.9	24	G2P1L0	37	Y	170/120	3		F,P,T					CS	P.CS	A	1.5	6,7	RD	Y
72	jaya	IE	low dose	28	Gr	HI	BO	27.5	5	G2P1L1	39	Y	180/120	4	H,BV						LN		A	2.75	7,8		
73	chithra	IE	low dose	22	XII	HI	BO		7	PRIMI	31		140/106	2	H,BV	F,P,I					LN		SB	2			
74	vijaya	IE	low dose	23	XII	HI	BO	25.4	5	PRIMI	40	Y	150/110	3	H	F,D,T				Y	LN		A	3			
75	devi	IE	low dose	29	X	HI	BO	24.5	6	G2P1L1	40		160/110	4	H	F,T					CS	FP	A	2.7	7,8		
76	vijaya	IE	low dose	25	X	HI	UB	23.9	10	PRIMI	40	Y	200/130	3	H,V,BV	F,P,I					CS	FD	A	2.5	7,8		
77	sivagami	IE	low dose	20	V	HI	BO	22.6	14	PRIMI	34		170/120	4		F,T					LN		A	2	6,8	IUGR	
78	maheswari	IE	low dose	20	V	HI	BO	26.2	6	PRIMI	38		170/110	2		F					CS	FP	A	3.6	7,8		

S.NO	NAME	TYPE OF ECLAMPSIA	TYPE OF REGIMEN	AGE	EDUCATION	RELIGION	BOOKING	BMI	STAY PERIOD	GRAVIDA	GEST AGE	ANTIHYPERTENSIVE IN PREGNANCY	ADMISSION BP	PROTENURIA	SYMPTOMS	SIDE EFFECTS	REGIMEN WITHHELD	RECURRENCE	POSTNATAL COMPLICATION	TREATMENT AT DISCHARGE	TYPE OF DELIVERY	INDICATION FOR CS	OUTCOME	BWT	APGAR	NEONATAL COMPLICATION	NICU ADMISSION
79	latha	IE	low dose	20	XII	HI	BO	29.9	5	G2P1L1	39		150/110	2		F,P,I					CS	P.CS	A	3.4	6,8		
80	maheswari	AP	low dose	30	V	HI	BO	25.3	15	G2P1L1	34	Y	160/100	3		F,P,I					LN		SB	1.6			
81	sahaya mary	AP	low dose	23	II	HI	BO	18.52	9	PRIMI	26		160/110	3		F,D,T					LN		SB	1.6			
82	deivayani	AP	low dose	20	VIII	HI	BO	23.5	25	PRIMI	34		130/80	3							CS	Abruption	A	2.2	,6,8		
83	selvabharathy	AP	low dose	29	V	HI	UB	20.9	13	G2P1L1	34		160/110	3		F,P,M	CONVULSION				LN		A	1.9	6,7	Hypotonia	Y
84	madhavi	AP	low dose	26	X	HI	BO		9	G2P1L1	27		140/90	4							LN		SB	0.8			
85	asmath	AP	low dose	21	XII	M	BO	20	7	G3P2L2	39		150/110	3		F,P,T					LN		A	2.75	7,8		
86	chithra	AP	low dose	19	XII	HI	UB		8	PRIMI	38		160/110	3		F,P					CS	CX	A	3.3	7,8		
87	selvarani	AP	low dose	24	V	HI	BI	23.3	11	G2P1L1	36		130/104	4	H						LN		A	1.6	6,7		
88	mahalakshmi	AP	low dose	22	Gr	HI	BO	29.5	8	PRIMI	38	Y	140/130	3		F,V,T					LN		SB	1.5			
89	kowsalya	AP	low dose	19	XII	HI	UB	24.8	8	PRIMI	36		140/110	2	H,V,BV			VENTILATOR			CS	DMC	A	2..75	7,8		
90	nandhini	AP	low dose	21	V	HI	BO	25.3	13	PRIMI	31		150/100	3		F,P,T					CS	FP	SB	1.2			
91	saraswathy	AP	low dose	25	XII	HI	BO	24.4	13	G3P2L2	32	Y	150/110	3		F,P,M,I	↓UO		RF		LN		A	1.6	7,8	IUGR	Y
92	malar	AP	low dose	21	XII	HI	BO	22.9	8	PRIMI	28		160/100	3							LN		A	1.4	6,7	hypotonia	Y
93	nirmala	AP	low dose	25	X	HI	BO	25.4	17	PRIMI	36		150/100	3							LN		SB	O.9			
94	selvi	AP	low dose	24	Gr	HI	BO	23.8	15	PRIMI	32		190/130	3		F,V					LN		A	2.3	7,8		
95	ankaleshwari	AP	low dose	20	II	HI	UB	22.5	12	PRIMI	38		170/100	4		F,P,I					CS	FP	E	1.4	3,6	hypotonia,RD	Y
96	deedomal	AP	low dose	22	II	HI	UB	20.4	10	PRIMI	38		140/110	4				VENTILATOR			CS	DMC	A	2.95	7,8		
97	satya	AP	low dose	23	UE	HI	UB		12	PRIMI	36		150/110	3	H,↓UO	F,P					CS	FD	A	2.2	3,6		
98	praveena	PP	low dose	21	II	HI	UB	24.2	5	P1L1		Y	150/110	3						Y	LN		A	2.8	7,8		
99	janaki	PP	low dose	22	II	HI	UB	26.1	9	P1L1		Y	150/110	4							CS		A	3.5	7,8		
100	deepa	PP	low dose	19	XII	HI	BO	25	5	P1L1		Y	140/110	3		F,P,M	CONVULSION				LN		A	3.2	7,8		

Note :

	IE	minent eclampsia	BO	ked outside	E	pigastric pain	I	induration	CS	arean section
	AP	epartum eclampsia	BI	oked IOG	↓UO	ased urine output	Ab	abscess	LN	our naturale
	PP	tpartum eclampsia	UB	BOOKED	F	flushing	T	thirst	DMC	maternal condition
	HI	hindu	H	eadache	D	ness/drowsiness	A	alive	FI	ed induction
	M	muslim	V	omitting	P	pain	DB	dead born	FP	e to progress
	C	christian	BV	ing of vision	M	iscle weakness	RD	iratory distress	CX	ourable cervix
	UE	uneducated	N	nausea	Y	yes	DTR	tendon reflex	FD	tal distress
	Gr	graduate	P	para	BP	lood presurre	RF	enal failure	P.CS	evious Iscs
	G	gravida	L	ive birth	A	abortion	CVT	l vein thrombosis	PPH	um haemorrhage
COLUMN 13/ 21	Y	hypertensive given							E	expired
COLUMN 28	Y	NICU admission							MRP	moval of placenta